

Contents

EYE..... 4

Q: Describe the characteristic changes observed in fundus examination of a child with i) Chronic Hypertension ii) Acute Lymphoblastic leukemia iii) Long standing diabetes mellitus 4

Q: Describe fundus findings of common pediatric diseases; add a note on cherry red spot 5

Q: Ophthalmic findings in IEM (inborn errors of metabolism) 8

Q: Enlist the acquired eye diseases in children ; Common causes, clinical features and management of acquired eye diseases in children 10

Q: Describe the various age-appropriate tests for checking visual acuity in children and their limitations 11

Q: What are the common causes of Blindness in children? Discuss steps to prevent Blindness in this group 13

Q: Congenital cataract: etiology; diagnosis; management and prevention..... 15

Q: Screening, classification and prevention of retinopathy of prematurity 21

ENT..... 30

Q: Outline development of normal hearing in children. List causes of Hearing impairment in a 1 year old child and its diagnostic approach 30

Q: Describe the types of hearing loss in children. Enumerate the causes of hearing loss in children. 31

Q: Describe the etiopathogenesis and clinical features in a 3 year old child with hearing impairment. What are the laboratory tests for assessment of such a child?	36
Q: Universal Neonatal Hearing Screening (UNHS).....	38
Q: Pediatric Cochlear Implant.....	39
Q: Acute Suppurative Otitis Media (ASOM) in Children; Outline the etiopathogenesis of acute suppurative otitis media. Discuss in brief the treatment and complications of acute suppurative otitis media (ASOM) in children.	40
Q: Recurrent Acute Otitis Media (RAOM) in Children	42
Q: Investigations and Treatment of Childhood Vertigo	43
SKIN	46
Q: Clinical conditions associated with Maculopapular rashes in children and their differential diagnosis.....	46
Q: Diagnosis and management of a 2-year-old child with Petechial skin rash	47
Q: Approach to a Case of Exanthematous Fever in a 14-Month-Old Child	50
Q: Differential Diagnosis for Bullous Skin Eruptions in Newborn Babies	52
Q: Enumerate the causes of persistent fever which are not due to infection. Describe the clinical presentation of ectodermal dysplasias.	53
Q: Scabies in Children.....	55
Q: Management of Vascular Nevi in Children	59
Q: Stevens-Johnson Syndrome (SJS)	61
Q: Erythema Multiforme.....	64

Q: Molluscum Contagiosum	65
Q: Seborrheic Dermatitis in Children and Neonates	67
Q: Erythema Nodosum	69
Q: Trichotillomania	70
Q: Pediculosis	72
Q: Acne in Adolescence	73
Q: Treatment of acne vulgaris	76
Q: Psychological Complications of Acne in Children	80
BONE AND JOINT DISORDERS	83
Q: Approach to a child with Talipes equinovarus at different ages (CTEV)	83
Q: Congenital Dislocation of Hip / DDH; Describe developmental dysplasia of the hip (DDH). Enumerate risk factors for the same. Enlist its clinical features. How do you confirm its diagnosis? Outline the management.	85
Q: Achondroplasia	88
Q: Classification and management of Osteogenesis Imperfecta	90

EYE

Q: Describe the characteristic changes observed in fundus examination of a child with i) Chronic Hypertension ii) Acute Lymphoblastic leukemia iii) Long standing diabetes mellitus

i) Chronic Hypertension

- **Retinal Arteriolar Narrowing:** Constriction of the small arteries in the retina.
- **Arteriovenous Nicking:** Arteries crossing veins, leading to vein constriction.
- **Cotton-Wool Spots:** White, fluffy lesions caused by nerve fiber layer infarcts.
- **Hemorrhages:** Flame-shaped or dot-blot hemorrhages may be present.
- **Optic Disc Edema:** Swelling of the optic disc may occur in severe cases.

Staging of Hypertensive Retinopathy

Stage 1: Mild Hypertensive Retinopathy

- **Retinal Arteriolar Narrowing:** Generalized or focal constriction of arterioles.

Stage 2: Moderate Hypertensive Retinopathy

- **Arteriovenous Nicking:** Arteriolar wall changes leading to compression of the vein at arteriovenous crossings.

Stage 3: Severe Hypertensive Retinopathy

- **Hemorrhages and Exudates:** Flame-shaped or dot-blot hemorrhages along with cotton-wool spots.
- **Microaneurysms:** Small outpouchings in the retinal capillaries.

Stage 4: Malignant Hypertensive Retinopathy

- **Optic Disc Edema:** Swelling of the optic disc.
- **Macular Star:** Exudates arranged in a star pattern around the macula.

ii) Acute Lymphoblastic Leukemia (ALL)

- **Roth Spots:** White-centered hemorrhages, indicative of retinal infiltrates.
- **Retinal Hemorrhages:** Dot-blot or flame-shaped, often multiple and bilateral.
- **Optic Nerve Infiltration:** Pale, swollen optic disc due to leukemic cells.
- **Retinal Vein Occlusion:** Due to hyperviscosity or direct leukemic infiltration.
- **Choroidal Lesions:** Creamy white lesions due to leukemic infiltrates.

iii) Long-Standing Diabetes Mellitus

- **Microaneurysms:** Small, red dots due to capillary wall outpouching.
- **Dot and Blot Hemorrhages:** Deep retinal hemorrhages.
- **Hard Exudates:** Yellow-white deposits from lipoprotein leakage.
- **Macular Edema:** Thickening of the macula, may cause vision loss.
- **Neovascularization:** New, fragile blood vessels forming on the retina or optic disc.

Staging of Diabetic Retinopathy

Stage 1: Non-Proliferative Diabetic Retinopathy (NPDR) - Mild

- **Microaneurysms:** The earliest clinical sign, small red dots on the retina.

Stage 2: NPDR - Moderate

- **Dot and Blot Hemorrhages:** Deep retinal hemorrhages.
- **Hard Exudates:** Lipid residues of serous leakage from damaged capillaries.

Stage 3: NPDR - Severe

- **Intraretinal Microvascular Abnormalities (IRMA):** Abnormal blood vessel growth.
- **Venous Beading:** Swelling of retinal veins.

Stage 4: Proliferative Diabetic Retinopathy (PDR)

- **Neovascularization:** New blood vessel formation on the retina or optic disc.
- **Vitreous Hemorrhage:** Bleeding into the vitreous cavity.
- **Tractional Retinal Detachment:** Retinal detachment due to fibrovascular proliferation.

-----X-----X-----

Q: Describe fundus findings of common pediatric diseases; add a note on cherry red spot

1. Detection of Ocular Diseases:

- **Retinopathy of Prematurity (ROP):** Critical in premature infants for early detection and management.
- **Congenital Cataracts:** Identification of lens opacities.
- **Retinal Dystrophies:** Early signs of inherited retinal disorders.

2. Identification of Systemic Diseases:

- **Diabetic Retinopathy:** In children with diabetes, early detection can prevent progression.
- **Hypertensive Retinopathy:** Can indicate underlying hypertension.
- **Infectious Diseases:** E.g., chorioretinitis in congenital infections like toxoplasmosis.

3. **Neurological Disorders:**

- **Papilledema:** Swelling of the optic disc indicating raised intracranial pressure.
- **Optic Atrophy:** Can be a sign of neurological conditions or previous optic neuritis.
- **Cranial Nerve Palsies:** Manifesting as abnormal ocular movements.

4. **Genetic Syndromes:**

- **Down Syndrome:** Associated with various ocular abnormalities.
- **Marfan Syndrome:** Lens dislocation and other ocular features.
- **Neurofibromatosis:** Lisch nodules and optic gliomas.

5. **Trauma Assessment:**

- **Retinal Hemorrhages:** Can indicate non-accidental injury (abuse) or severe head trauma.
- **Comotio Retinae:** Retinal changes post blunt trauma.

6. **Screening for Amblyopia and Strabismus:**

- **Early Detection:** Crucial for timely intervention to prevent permanent vision loss.

7. **Monitoring of Treatment:**

- **Post-Surgical Evaluation:** E.g., after cataract or strabismus surgery.
- **Response to Systemic Treatment:** In conditions like diabetes or hypertension.

8. **Educational and Developmental Implications:**

- **Visual Impairments:** Can affect learning and development, requiring early intervention.

Disease/Condition	Fundus examination findings	Clinical Relevance
<i>Chronic Hypertension</i>	Arteriolar-narrowing Hemorrhages Exudates Papilledema	Indicates target organ damage, may require antihypertensive therapy
<i>Acute Lymphoblastic Leukemia</i>	Roth spots Retinal hemorrhages Cotton wool spots	May indicate leukemic infiltration or hyperviscosity
<i>Long-standing Diabetes Mellitus</i>	Microaneurysms Retinal hemorrhages Hard exudates Neovascularization	Indicates diabetic retinopathy, may require laser photocoagulation or anti-VEGF therapy
<i>Optic Neuritis</i>	Optic disc swelling Peripapillary hemorrhages	Suggestive of demyelinating disease, may require corticosteroid treatment
<i>Leber's Hereditary Optic Neuropathy - LHON</i>	Optic disc pallor Vascular tortuosity	Mitochondrial disorder leading to vision loss, often bilateral
<i>Pseudotumor Cerebri</i>	Papilledema, Venous engorgement	Indicates raised intracranial pressure, may require lumbar puncture or shunt
<i>Tuberous Sclerosis</i>	Retinal astrocytic hamartomas "mulberry" lesions	Associated with multi-system tumors, may indicate CNS involvement
<i>Neurofibromatosis Type 1- NF1</i>	Optic nerve gliomas Lisch nodules	May indicate CNS tumors, requires neuroimaging
<i>Sturge-Weber Syndrome</i>	Diffuse choroidal hemangioma "tomato ketchup" fundus	Vascular malformation, may be associated with glaucoma
<i>Alpers' Disease</i>	Optic atrophy Retinal degeneration	Progressive neurodegenerative disease, poor prognosis
<i>Tay-Sachs Disease</i>	Cherry-red spot at the macula	Indicates lysosomal storage disease, associated with neurodegeneration

<i>Krabbe Disease</i>	Optic atrophy "salt and pepper" retinopathy	Leukodystrophy, associated with rapid neurodegeneration
<i>Batten Disease</i>	Retinal degeneration Optic disc pallor	Progressive neurodegenerative disorder, vision loss is common
<i>Mitochondrial Disorders</i>	Pigmentary retinopathy Optic atrophy	Systemic disease, may have stroke-like episodes and seizures
<i>Acute Disseminated Encephalomyelitis (ADEM)</i>	Optic disc edema Retinal hemorrhages	Post-infectious or post-vaccination, may require immunomodulatory treatment

Cherry-Red Spot:

- Cherry-red spot is a classic fundus finding seen in certain lysosomal storage diseases like Tay-Sachs, Niemann-Pick, and others
- It is caused by the accumulation of storage material in the retinal ganglion cells, which makes the surrounding retina appear pale, accentuating the normal red color of the fovea
- This is a hallmark of severe systemic disease and is usually associated with progressive neurodegeneration.

-----X-----X-----

Q: Ophthalmic findings in IEM (inborn errors of metabolism)

(Note this question is asking ophthalmic findings in general; so you should not restrict your answer to fundus findings only)

Inborn Error of Metabolism	Ophthalmic findings	Clinical Relevance
<i>Tay-Sachs Disease</i>	Cherry-red spot at the macula	Indicates lysosomal storage disease, associated with neurodegeneration
<i>Niemann-Pick Disease</i>	Cherry-red spot Corneal clouding	Suggests sphingomyelin accumulation, systemic involvement likely
<i>Gaucher Disease</i>	Pingueculae Retinal infiltrates	Indicates glucocerebroside accumulation, may have systemic symptoms

<i>Fabry Disease</i>	Corneal verticillata Cataract	Indicates glycosphingolipid accumulation, may have renal/cardiac involvement
<i>Mucopolysaccharidoses</i>	Corneal clouding Retinal degeneration	Indicates glycosaminoglycan accumulation, systemic involvement common
<i>Alkaptonuria</i>	Blue-black discoloration of sclera	Indicates homogentisic acid accumulation, may have joint involvement
<i>Wilson Disease</i>	Kayser-Fleischer rings Sunflower cataract	Indicates copper accumulation, may have hepatic/neurologic symptoms
<i>Homocystinuria</i>	Ectopia lentis Myopia Retinal detachment	Indicates homocysteine accumulation, may have systemic vascular issues
<i>Galactosemia</i>	Cataracts Pseudotumor cerebri	Indicates galactose accumulation, may have liver dysfunction
<i>Refsum Disease</i>	Retinitis pigmentosa Optic atrophy	Indicates phytanic acid accumulation, may have neurologic symptoms
<i>Oculocutaneous Albinism</i>	Nystagmus Iris transillumination	Indicates melanin synthesis defect, may have skin/hair involvement
<i>Congenital Hyperammonemia</i>	Papilledema Optic atrophy	Indicates ammonia accumulation, may have neurologic symptoms
<i>Maple Syrup Urine Disease</i>	Optic atrophy Retinal degeneration	Indicates branched-chain amino acid accumulation, may have neurologic symptoms
<i>Zellweger Syndrome</i>	Cataracts Retinal degeneration	Indicates peroxisomal disorder, associated with severe neurologic symptoms
<i>Cystinosis</i>	Corneal crystals Retinopathy	Indicates cystine accumulation, may have renal dysfunction

-----X-----X-----

Q: Enlist the acquired eye diseases in children ; Common causes, clinical features and management of acquired eye diseases in children

Acquired Eye Disease	Common Causes	Clinical Features	Management
Conjunctivitis	Viral, bacterial, allergic	Redness, discharge, itching	Antibiotics for bacterial, antihistamines for allergic, supportive for viral; Avoid packing of eyes
Stye (Hordeolum)	Staphylococcal infection	Painful lump on eyelid	Warm compress, topical antibiotics
Chalazion	Blocked meibomian gland	Painless lump on eyelid	Warm compress, corticosteroid injections, surgical removal
Blepharitis	Bacterial, seborrheic	Itchy, red eyelids, crust formation	Lid hygiene, topical antibiotics, lubricating eye drops
Keratitis	Infection, trauma	Pain, photophobia, blurred vision	Antibiotics, antifungals, or antivirals depending on cause
Uveitis	Autoimmune, infection	Pain, redness, blurred vision	Corticosteroids, immunosuppressants, treat underlying cause
Retinoblastoma	Genetic mutations	White pupil, strabismus	Chemotherapy, radiation, enucleation
Amblyopia	Strabismus, refractive errors	Reduced vision in one eye	Patching stronger eye, corrective lenses
Strabismus	Muscle imbalance	Misaligned eyes	Glasses, patching, surgery
Refractive Errors	Prematurity; Idiopathic	Blurred vision	Corrective lenses, refractive surgery in older children
Retinal Detachment	Trauma, myopia	Sudden vision loss, floaters	Urgent surgical intervention

Optic Neuritis	Autoimmune, infection	Pain, vision loss	Corticosteroids, treat underlying cause
Ocular Trauma	Physical injury	Pain, redness, vision loss	Emergency treatment, may require surgery
Glaucoma	Increased intraocular pressure	Pain, redness, vision loss	Medication to lower pressure, surgery
Cataract	Trauma, metabolic disorders	Cloudy vision	Surgical removal of cataract

-----X-----X-----

Q: Describe the various age-appropriate tests for checking visual acuity in children and their limitations

Age Group	Test for Visual Acuity	Description	Limitations
Newborn-3 months	Fixate and Follow	Assess if the infant can fixate on an object and follow it with their eyes.	Not a quantitative measure of visual acuity; only assesses gross visual behavior.
3-6 months	Teller Acuity Cards	Grating acuity cards are shown to assess the finest grating the infant can resolve.	Requires specialized cards; not a direct measure of letter acuity.
6-12 months	Preferential Looking (PL)	Utilizes the infant's natural tendency to look at more visually interesting stimuli.	Subjective; dependent on infant's attention and cooperation.
1-3 years	Lea Symbols or HOTV	Child matches or names simple symbols or letters.	Requires some level of understanding and cooperation from the child.
3-5 years	Snellen E Chart	Child is asked to identify the	Requires understanding of directions; not suitable for

		orientation of the letter "E" (up, down, left, right).	illiterate or non-English speaking kids; for them U shapes can be useful alternative.
5+ years	Snellen Chart	Traditional chart with letters that decrease in size.	Requires literacy and understanding of the task; may not be suitable for children with special needs.
Any age	Retinoscopy	Objective method where light is shone into the eye to assess refractive error.	Does not measure visual acuity directly; requires specialized equipment and expertise.
Any age	Autorefractors	Automated machine that estimates refractive error.	Not a direct measure of visual acuity; calibration and machine error can affect results.

Methods for Testing Near Vision in Children

Method	Description	Age Group	Limitations
Lea Symbols Near Card	Utilizes symbols like a circle, square, apple, or house for matching or naming. The card is held at a distance of 16 inches (40 cm) from the child.	2-5 years	Requires some level of understanding and cooperation from the child.
Jaeger Chart	A handheld card with paragraphs of text in various font sizes, held at a reading distance (usually 14 inches).	5+ years	Requires literacy and understanding of the task; not suitable for pre-literate children.
Rosenbaum Pocket Vision Screener	A pocket-sized card with numbers or letters, held at a distance of 14 inches.	5+ years	Requires literacy; not suitable for younger children.
Near Point of Convergence (NPC)	Measures the closest point at which eyes can converge comfortably. A small target is moved towards the child until one eye deviates outward.	5+ years	Requires cooperation and understanding of the task; not suitable for very young children.

Accommodation Tests	Measures the eye's ability to focus on near objects. Often done using retinoscopy or dynamic retinoscopy.	Any age	Requires specialized equipment and expertise; not a direct measure of near visual acuity.
---------------------	---	---------	---

Note:

- For younger children or those with developmental delays, near vision may be assessed more qualitatively through observation of how the child interacts with close-up objects or tasks.
- Near vision problems could be a sign of other issues like convergence insufficiency, accommodative dysfunction, or even systemic conditions like hyperopia.

-----X-----X-----

Q: What are the common causes of Blindness in children? Discuss steps to prevent Blindness in this group

Common Causes of Blindness in Children:

Cause	Description
Congenital Cataract	Clouding of the lens present at birth or developing shortly thereafter.
Retinopathy of Prematurity - ROP	Abnormal blood vessel development in the retina, often seen in premature infants.
Optic Nerve Hypoplasia	Underdevelopment of the optic nerve, often associated with other systemic issues.
Glaucoma	Increased intraocular pressure leading to optic nerve damage; can be congenital or acquired.
Ocular Trauma	Injury to the eye that may result in vision loss.
Infections	Ocular infections like congenital rubella syndrome, toxoplasmosis, or bacterial meningitis.
Leber's Congenital Amaurosis	A group of inherited retinal diseases causing severe vision loss at birth or in the first months.

Vitamin A Deficiency	A preventable cause of blindness, common in developing countries.
Inborn Errors of Metabolism	Rare genetic disorders that can affect vision, such as Tay-Sachs disease.

Steps to Prevent Blindness in Children

1. **Prenatal Care:** Regular prenatal check-ups to screen for infections that could affect the fetus.
2. **Newborn Screening:** Early detection of congenital issues through newborn eye screening.
3. **Vaccination:** Timely vaccination against diseases like measles, which can cause blindness.
4. **Nutrition:** Adequate intake of Vitamin A and other essential nutrients to prevent nutritional deficiencies.
5. **Regular Eye Exams:** Routine eye check-ups for early detection of refractive errors, strabismus, or other issues.
6. **Early Intervention:** Prompt treatment for conditions like cataract, glaucoma, or retinopathy of prematurity which requires laser treatment.
7. **Public Awareness:** Educating parents and caregivers about the importance of eye health and regular screenings.
8. **Safety Measures:** Use of protective eyewear during sports and other high-risk activities to prevent trauma.
9. **Genetic Counseling:** For families with a history of hereditary eye diseases.
10. **Access to Care:** Ensuring that children, especially those in underserved areas, have access to quality eye care.
11. **Special Education:** For children with irreversible vision loss, early intervention programs to facilitate development.
12. **Hygiene:** Educating about the importance of eye hygiene can prevent many infections.

By implementing these preventive measures, the risk of childhood blindness can be significantly reduced.

-----X-----X-----

Q: Congenital cataract: etiology; diagnosis; management and prevention

Causes of Congenital Cataract

Cause Category	Specific Causes
Genetic Factors	Autosomal dominant, autosomal recessive, or X-linked inheritance patterns.
Metabolic Disorders	Galactosemia, hypocalcemia, and other inborn errors of metabolism.
Intrauterine Infections	Rubella, cytomegalovirus (CMV), syphilis, and toxoplasmosis.
Trauma	Direct injury to the eye or head during childbirth or due to prenatal factors.
Drug Exposure	Maternal use of corticosteroids, antiepileptic drugs, or other teratogenic substances during pregnancy.
Associated Syndromes	Down syndrome, Marfan syndrome, and other systemic or syndromic conditions.
Idiopathic	Unknown causes; no identifiable risk factors.

Diagnosis of Congenital Cataract

1. **Clinical Examination:** A thorough eye examination, including the red reflex test, is usually the first step in diagnosis.
2. **Slit-Lamp Examination:** To visualize the lens opacity and other anterior segment abnormalities.
3. **Retinoscopy:** To assess the refractive error and the impact of the cataract on vision.
4. **Ultrasound Biomicroscopy (UBM):** To evaluate the lens and its relationship with other anterior segment structures, especially if the cataract is dense.
5. **Genetic Testing:** Particularly if other family members are affected or if the cataract is syndromic.
6. **Metabolic Screening:** For conditions like galactosemia, especially if other systemic symptoms are present.

7. **Imaging:** MRI or CT scans may be necessary if other ocular or systemic abnormalities are suspected.

Management of Congenital Cataract

Surgical Management

1. **Cataract Extraction:**

- **Timing:** Surgery is usually performed as early as possible to prevent amblyopia.
- **Method:** Phacoemulsification or aspiration techniques are commonly used.

2. **Intraocular Lens (IOL) Implantation:**

- **Type:** Single-piece acrylic IOLs are commonly used.
- **Power:** Calculated based on biometry and expected postoperative growth of the eye.

3. **Postoperative Care:**

- **Medication:** Topical antibiotics and steroids.
- **Follow-up:** Regular follow-ups to monitor for complications like glaucoma and posterior capsular opacification.

Non-Surgical Management

1. **Optical Correction:**

- **Contact Lenses:** Used if surgery is deferred or contraindicated.
- **Glasses:** For residual refractive errors post-surgery.

2. **Amblyopia Treatment:**

- **Occlusion Therapy:** Patching the better eye to stimulate the affected eye.
- **Pharmacologic Penalization:** Using atropine drops in the better eye.

3. **Low Vision Aids:** For children with irreversible vision loss.

Systemic Management

1. **Genetic Counseling:** For families with a history of congenital cataracts.
2. **Metabolic Control:** In cases where the cataract is secondary to a metabolic disorder.

Monitoring and Follow-up

1. **Regular Eye Examinations:** To monitor for complications and assess visual development.
2. **Rehabilitation Services:** Including occupational and educational support.

Prevention of Congenital Cataract:

1. **Prenatal Screening:** Regular prenatal check-ups to identify and manage maternal infections and metabolic disorders.
2. **Genetic Counseling:** For couples with a family history of congenital cataracts or other hereditary conditions that may cause cataracts.
3. **Vaccination:** Ensuring that the mother is vaccinated against diseases like rubella that can cause congenital cataracts. However vaccination during pregnancy is contraindicated.
4. **Medication Review:** Assessing and modifying the medications that a pregnant woman is taking, especially if they are known to be associated with congenital cataracts.
5. **Nutritional Supplementation:** Adequate intake of essential nutrients and vitamins, such as Vitamin C and E, which are antioxidants that may play a role in preventing cataracts.
6. **Avoid Alcohol and Smoking:** As Both of these can increase the risk of congenital cataracts, this advice to be given preconceptionally during counselling as maximum damage occurs before woman is aware of her pregnancy.
7. **Control of Metabolic Disorders:** Effective management of diabetes and other metabolic conditions in the mother can reduce the risk.
8. **Safe Childbirth Practices:** Minimizing the risk of trauma during childbirth by opting for safe delivery methods.
9. **Newborn Screening:** Early detection through newborn eye examinations can lead to timely intervention, reducing the impact.
10. **Public Awareness:** Educating the public about the risk factors and preventive measures for congenital cataracts.

-----X-----X-----

Q: Ophthalmia Neonatorum

- ✚ Ophthalmia Neonatorum is a preventable and treatable condition, but it requires prompt recognition and appropriate management to prevent serious complications.
- ✚ Ongoing efforts in public health, maternal care, and neonatal management are crucial to reduce the incidence and impact of this condition.

Definition

- **Ophthalmia Neonatorum:** A form of conjunctivitis occurring in newborns within the first month of life. It's a significant cause of neonatal morbidity and potential corneal impairment, endophthalmitis and systemic dissemination.

Etiology

- **Infectious Causes:**
 - **Bacterial:** Neisseria gonorrhoeae, Chlamydia trachomatis, Staphylococcus aureus, Streptococcus pneumoniae, and others.
 - **Viral:** Herpes Simplex Virus (HSV).
- **Non-Infectious Causes:**
 - Chemical irritation from prophylactic eye drops.
 - Blocked tear duct.

Clinical Presentation

- Redness and swelling of the conjunctiva.
- Eyelid edema.
- Purulent or watery discharge.
- Photophobia in some cases.

Diagnosis

- **Clinical Assessment:** History of maternal infections, timing of symptom onset.
- **Laboratory Tests:**
 - Swab of conjunctival discharge for Gram stain, culture, and sensitivity.
 - PCR for Chlamydia trachomatis and HSV.

- **Differential Diagnosis:** Distinguish from other causes of neonatal conjunctivitis, including chemical conjunctivitis and blocked tear duct.

Management

- **Immediate Empirical Treatment:**
 - Broad-spectrum topical antibiotics (e.g., erythromycin, tetracycline) while awaiting culture results.
 - Systemic antibiotics in cases of gonococcal or chlamydial infection.
- **Specific Treatment:**
 - Gonococcal infection: Intravenous or intramuscular ceftriaxone.
 - Chlamydial infection: Oral erythromycin.
 - HSV infection: Systemic acyclovir.
- **Supportive Care:**
 - Saline eye washes to remove discharge.
 - Warm compresses to reduce eyelid edema.

Causes	Diagnostic Approach	Management
<i>Neisseria gonorrhoeae</i>	Gram stain and culture of conjunctival discharge PCR for gonococcus	Systemic antibiotics: Intravenous or intramuscular ceftriaxone Topical antibiotics: Erythromycin or tetracycline ointment
<i>Chlamydia trachomatis</i>	PCR for Chlamydia Conjunctival discharge culture	Oral erythromycin Topical erythromycin or tetracycline ointment
<i>Herpes Simplex Virus (HSV)</i>	PCR for HSV Viral culture of eye discharge	Systemic acyclovir Ophthalmology consultation for potential topical antiviral therapy
<i>Staphylococcus aureus</i>	Culture and sensitivity of discharge Gram stain	Topical antibiotics based on sensitivity (e.g., erythromycin)
<i>Streptococcus pneumoniae</i>	Culture and sensitivity Gram stain	Topical antibiotics based on culture results
<i>Chemical Irritation</i> (e.g., from kajal, wax and other traditional, antibiotic drops)	Clinical diagnosis based on history and timing post prophylaxis	Supportive care: Saline eye washes Typically self-limiting

Blocked Tear Duct	Clinical assessment Exclusion of infectious causes	Gentle massage of the lacrimal sac Monitoring for resolution or signs of secondary infection
--------------------------	---	---

- **Empirical Treatment:** Initiate broad-spectrum topical antibiotics while awaiting culture results.
- **Supportive Care:** Includes saline eye washes for all types to remove discharge and warm compresses for eyelid edema.
- **Prevention:** Maternal screening and treatment for STDs, prophylactic eye ointment (erythromycin or tetracycline) at birth.
- **Follow-up:** Essential for all cases to monitor response to treatment and for any complications.
- **Referral:** Severe cases or those with complications should be referred to a pediatric ophthalmologist.

Prevention

- **Maternal Screening and Treatment:** Screening and treating pregnant women for STDs.
- **Prophylaxis at Birth:**
 - Erythromycin or tetracycline ointment in the eyes of all newborns as a preventive measure.
 - Silver nitrate drops, though less commonly used due to potential chemical irritation.

Complications

- Corneal ulceration and scarring.
- Potential blindness if untreated, especially in gonococcal infections.
- Systemic spread of infection in some cases.

Follow-up and Monitoring

- Close follow-up to monitor response to treatment.
- Referral to a pediatric ophthalmologist for severe cases or complications.
- Monitoring for signs of systemic infection.

Public Health Implications

- Reporting of cases to public health authorities, especially for gonococcal and chlamydial infections.

- Education of healthcare providers and parents about prevention and early recognition.

Research and Future Directions

- Development of more effective prophylactic measures.
- Studies on the long-term outcomes of affected infants.
- Exploration of novel diagnostic and therapeutic approaches.

-----X-----X-----

Q: Screening, classification and prevention of retinopathy of prematurity

Screening for Retinopathy of Prematurity (ROP)

Criteria for Screening	Western NICUs	Current Practice guidelines in INDIA
Birth Weight	≤ 1500 grams	≤ 2000 grams
Gestational Age	≤ 30 weeks	≤ 34 weeks
Clinical Instability	Unstable clinical course High-Risk Factors: Among Infants with a stable birth weight and gestational age but having risk factors like prolonged oxygen therapy, sepsis, multiple blood transfusions, other intensive care, etc.	

Timing: First screening should be done between 31-33 weeks postmenstrual age or 4-6 weeks after birth, whichever is later.

However for certain subgroup of babies who had stormy course on ventilation; screening as early as 2 to 3 weeks postnatal age is advised in Indian population because of their peculiar susceptibility to APROP

Frequency: Weekly or bi-weekly, depending on the severity and zone of the disease.

Method: Indirect ophthalmoscopy with scleral depression.

Screening Personnel

- Screening should be conducted by ophthalmologists trained in the identification of ROP.

Screening Method

- Indirect ophthalmoscopy is the standard method for ROP screening in India.

Follow-up and Treatment

- Infants with any stage of ROP in Zone I, Stage 3 or worse in Zone II, or "Plus Disease" should be considered for immediate treatment.
- Treatment options include laser photocoagulation, cryotherapy, and more recently, intravitreal injections of anti-VEGF agents.

Parental Counseling

- Parents should be educated about the importance of timely screening and follow-up visits.

Documentation

- Detailed documentation of findings and any treatment administered should be maintained for each infant screened.

Classification of ROP

Stage	Description
Stage 1	Demarcation line separating vascularized and avascular retina
Stage 2	Elevated ridge with volume
Stage 3	Extraretinal fibrovascular proliferation
Stage 4	Partial retinal detachment
Stage 5	Total retinal detachment

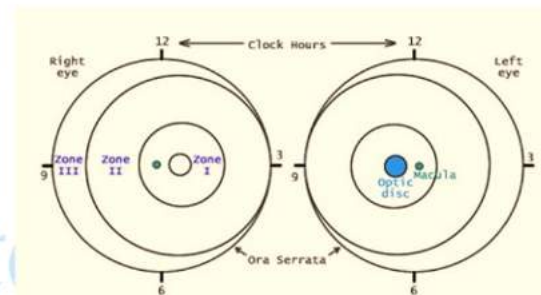
Detailed Staging:

Stage	Description	Sub-stage A	Sub-stage B
0	Immature or flat vascularization	No evidence of ROP	Immature retinal vessels
1	Demarcation line	Thin line separating avascular retina from posterior retina	Thick line with structure

2	Ridge with volume	Ridge without extraretinal fibrovascular proliferation	Ridge with extraretinal fibrovascular proliferation
3	Ridge with extraretinal fibrovascular proliferation	Extraretinal fibrovascular proliferation less than 50% of the ridge	Extraretinal fibrovascular proliferation more than 50% of the ridge
4	Subtotal retinal detachment	Extrafoveal detachment	Foveal detachment but with some macular attachment
5	Total retinal detachment	Partially open funnel	Totally closed funnel

Zones of ROP

Zone	Description
Zone I	Centered on the optic disc, radius defined by twice the distance from the optic disc to the macula
Zone II	From the edge of Zone I to the ora serrata anteriorly
Zone III	Residual crescent of retina anterior to Zone II



Plus Disease: Defined as dilation and tortuosity of the retinal blood vessels, indicating disease severity.

Aggressive Posterior ROP (AP-ROP)

- Aggressive Posterior ROP is a severe form of ROP that occurs in the posterior retina, usually in Zone I or posterior Zone II.
- It is characterized by rapid progression and a higher risk of retinal detachment. AP-ROP often lacks the classic demarcation lines or ridges seen in other stages and may present with "plus disease."
- Immediate intervention is crucial, and it often requires more aggressive treatment strategies, such as laser photocoagulation or intravitreal anti-VEGF injections.

Prevention of ROP:

1. Oxygen Regulation:

- Maintain oxygen saturation in the target range to prevent hyperoxia.
- Continuous oxygen monitoring.

2. Nutritional Support:

- Early enteral feeding.
- Supplement with essential fatty acids and antioxidants.

3. Avoidance of Infection:

- Strict aseptic precautions.
- Prophylactic antibiotics as needed.

4. Blood Transfusions:

- To be done cautiously; avoid large volume rapid transfusions.

5. Parental Education:

- Educate about the importance of regular screening and follow-up.

6. Systemic Treatment:

- Use of medications like intravitreal anti-VEGF agents is still under study for prevention.

7. Regular Follow-up:

- Critical for timely intervention and prevention of blindness.

Early detection through rigorous screening, accurate classification, and timely intervention are key to the management and prevention of ROP.

-----X-----X-----

Q: Penetrating ocular trauma in toddler

- ❖ Approaching a case of penetrating ocular trauma in a toddler requires a detailed, systematic, and expert approach, considering both the immediate needs and long-term implications of the injury
- ❖ Managing penetrating ocular trauma in a toddler is a complex and challenging scenario that requires prompt, meticulous, and expert care
- ❖ The approach encompasses immediate stabilization, detailed ocular examination, appropriate imaging, surgical intervention, and long-term follow-up for rehabilitation and monitoring of complications

- ❖ The goal is to preserve as much visual function as possible while minimizing the risk of long-term sequelae
- ❖ The involvement of a multidisciplinary team and the support for the child and family are crucial components of comprehensive care.

Initial Assessment and Stabilization:

1. Triage and Primary Survey:

- Assess the child's overall stability (ABCs: Airway, Breathing, Circulation).
- Evaluate for polytrauma, particularly cranial or facial injuries.

2. Pain and Anxiety Management:

- Administer age-appropriate analgesics and anxiolytics.
- Consider the child's distress and the parents' anxiety in the management plan.

3. Ocular Examination:

- Avoid manipulation of the globe to prevent exacerbation of the injury.
- Use a penlight for a cursory external examination.
- Check for the presence of a foreign body, prolapsed uveal tissue, or a visible wound.

4. Protection of the Eye:

- Apply a rigid, non-compressive eye shield.
- Avoid any pressure dressings.

Detailed Ophthalmic Evaluation:

1. Visual Acuity Assessment:

- Attempt to assess visual acuity, if feasible, considering the child's age and cooperation.

2. Slit-lamp Biomicroscopy:

- Conduct a careful anterior segment examination.
- Look for corneal lacerations, anterior chamber depth changes, iris damage, or lens involvement.

3. Intraocular Pressure (IOP) Measurement:

- If the globe is intact, measure IOP gently, being cautious not to exert pressure on the globe.

4. **Fundus Examination:**

- Perform indirect ophthalmoscopy to assess the posterior segment, if the anterior segment permits.

Imaging:

1. **Orbital CT Scan:**

- Obtain a high-resolution CT scan to evaluate the extent of injury, detect intraocular or orbital foreign bodies, and assess for orbital fractures.

Surgical Management:

1. **Surgical Repair:**

- Plan for urgent surgical exploration and repair under general anesthesia.
- The primary goals are to restore the integrity of the globe, remove any intraocular foreign bodies, and repair any retinal or choroidal detachments.

2. **Microsurgical Techniques:**

- Employ microsurgical techniques for precise repair of lacerations.
- Consider the use of intraocular tamponades if needed.

3. **Antibiotic Prophylaxis:**

- Administer systemic broad-spectrum antibiotics to prevent endophthalmitis.

4. **Tetanus Prophylaxis:**

- Ensure tetanus prophylaxis is up to date, especially if a foreign body is involved.

Postoperative Care:

1. **Topical Medications:**

- Prescribe topical antibiotics and steroids to reduce inflammation and prevent infection.

2. **IOP Monitoring:**

- Regularly monitor intraocular pressure postoperatively.

3. **Follow-up Examinations:**

- Schedule frequent follow-up visits to monitor the healing process and detect early signs of complications.

Long-term Management and Rehabilitation:

1. **Visual Rehabilitation:**

- Assess and manage amblyopia, especially in very young children.
- Provide optical correction if necessary (e.g., aphakic glasses, contact lenses).

2. **Monitoring for Complications:**

- Be vigilant for late complications such as traumatic cataract, glaucoma, retinal detachment, or sympathetic ophthalmia.

3. **Family Counseling and Support:**

- Provide detailed counseling to the family regarding the prognosis, potential complications, and the importance of follow-up care.

4. **Multidisciplinary Approach:**

- Involve pediatricians, ophthalmologists, anesthetists, and possibly a child psychologist for comprehensive care.

-----X-----X-----

Q: Ocular manifestation of systemic disease

Systemic Disease Category	Specific Disease	Ocular Manifestations
Endocrine Disorders	Diabetes Mellitus	Diabetic retinopathy (Non-proliferative and proliferative) Diabetic macular edema Early onset cataract Cranial nerve palsies
	Thyroid Disorders (Graves' Disease)	Exophthalmos (proptosis) Eyelid retraction, lagophthalmos Extraocular muscle involvement Exposure keratopathy
Rheumatologic Disorders	Rheumatoid Arthritis	Keratoconjunctivitis sicca Episcleritis and scleritis Secondary Sjögren's syndrome

	Systemic Lupus Erythematosus (SLE)	Retinal vasculitis Cotton wool spots Optic neuropathy
	Ankylosing Spondylitis	Acute anterior uveitis
Infectious Diseases	HIV/AIDS	HIV retinopathy CMV retinitis Kaposi's sarcoma of the eyelid
	Syphilis	Interstitial keratitis Chorioretinitis Optic atrophy
	Tuberculosis	Choroidal tubercles Uveitis
Cardiovascular Diseases	Hypertension	Hypertensive retinopathy Retinal artery and vein occlusions
	Atherosclerosis	Retinal emboli Ischemic optic neuropathy
Hematologic Disorders	Sickle Cell Disease	Retinal vessel occlusions Proliferative sickle retinopathy Retinal detachment
	Leukemia	Retinal hemorrhages Leukemic infiltrates
Neurological Disorders	Multiple Sclerosis	Optic neuritis Internuclear ophthalmoplegia
	Myasthenia Gravis	Ptosis Ocular muscle weakness
Renal Disorders	Hypertensive Nephropathy	Similar to hypertensive retinopathy
	Chronic Renal Failure	Band keratopathy Calcific conjunctival deposits
Gastrointestinal Disorders	Crohn's Disease and Ulcerative Colitis	Episcleritis Uveitis
Dermatological Disorders	Psoriasis	Uveitis Conjunctivitis
	Rosacea	Rosacea keratitis Chalazion and blepharitis
Pediatric rheumatology Disorders	Juvenile Idiopathic Arthritis	Chronic anterior uveitis
Neonate	Retinopathy of Prematurity	In premature infants, related to oxygen therapy
Oncological Disorders	Paraneoplastic Syndromes	CAR (Cancer-associated retinopathy) MAR (Melanoma-associated retinopathy)

	Lymphoma	Intraocular lymphoma
--	----------	----------------------

---X-----X---



ENT

Q: Outline development of normal hearing in children. List causes of Hearing impairment in a 1 year old child and its diagnostic approach

Development of Normal Hearing in Children

- **Prenatal Period:**
 - Fetal hearing development begins around the 24th week of gestation.
 - Reacts to loud sounds by the third trimester.
- **Birth to 3 Months:**
 - Startle response to loud sounds.
 - Soothed by familiar voices.
- **3 to 6 Months:**
 - Turns eyes or head towards sound sources.
 - Enjoys musical toys and human voices.
- **6 to 9 Months:**
 - Responds to soft sounds and familiar voices.
 - Understands basic sound cues ("no," "bye-bye").
- **9 to 12 Months:**
 - Begins to mimic simple sounds and intonations.
 - Understands common words and simple directions.
- **1 to 2 Years:**
 - Vocabulary expands rapidly.
 - Begins to form simple sentences and follows simple commands.
- **2 to 3 Years:**
 - Understands more complex sentences.
 - Hears and understands nearly everything said at home or daycare.
- **3 to 5 Years:**
 - Vocabulary and sentence complexity continue to grow.

- Can carry on detailed conversations and understand most of what is said, even in noisy environments.

Causes of Hearing Impairment in a 1-Year-Old Child

Causes	Description
Congenital Factors	Genetic predisposition, prenatal exposure to infections like CMV
Otitis Media	Middle ear infection common in young children
Noise Exposure	Exposure to loud noises can damage the auditory nerve
Meningitis	Can cause sensorineural hearing loss
Ototoxic Medication	Some medications are toxic to auditory nerve or cochlea
Trauma	Head injuries can affect hearing
Syndromic Conditions	Conditions like Waardenburg syndrome, Usher syndrome

Diagnostic Approach

- **Initial Screening:** Universal newborn hearing screening before leaving the hospital.
- **Audiological Evaluation:** Comprehensive testing by an audiologist.
- **Tympanometry:** To assess middle ear function.
- **Otoacoustic Emissions (OAEs):** To check the inner ear's response to sound.
- **Auditory Brainstem Response (ABR):** To evaluate the auditory nerve.
- **Genetic Testing:** If a genetic cause is suspected.
- **Imaging Studies:** MRI or CT scans if structural abnormalities are suspected.

-----X-----X-----

Q: Describe the types of hearing loss in children. Enumerate the causes of hearing loss in children.

Types of Hearing Loss in Children

Type of Hearing Loss	Description	Affected Part of Ear	Common Causes
Conductive Hearing Loss	Difficulty in sound conduction from outer ear to inner ear	Outer or Middle Ear	Otitis media, earwax, foreign bodies
Sensorineural Hearing Loss	Damage to inner ear or auditory nerve	Inner Ear or Auditory Nerve	Genetic factors, meningitis, noise exposure
Mixed Hearing Loss	Combination of conductive and sensorineural	Both Outer/Middle and Inner Ear	Chronic ear infection along with inner ear damage
Auditory Neuropathy Spectrum Disorder	Sound enters the inner ear normally but transmission to the brain is impaired	Auditory Nerve	Prematurity, hyperbilirubinemia, anoxia

Causes of Hearing Loss in Children

Congenital Causes

- Genetic factors
- Maternal infections during pregnancy (e.g., rubella, CMV)
- Prematurity
- Low birth weight
- Anoxia (lack of oxygen) during birth

Acquired Causes

- Chronic ear infections (Otitis media)
- Meningitis
- Measles
- Encephalitis
- Noise exposure
- Ototoxic medications (e.g., aminoglycosides)
- Head trauma

- Tumors affecting the auditory pathway

Syndromic Conditions

- Waardenburg syndrome
- Usher syndrome
- Pendred syndrome
- Alport syndrome

Environmental Factors

- Exposure to loud noises
- Ototoxic chemicals

Other Medical Conditions

- Hyperbilirubinemia
- Autoimmune diseases
- Mumps
- **Michel's Aplasia:** This is a rare congenital condition where the inner ear is completely undeveloped. It results in profound sensorineural hearing loss and is often diagnosed shortly after birth.
- **Mondini's Aplasia:** This is a congenital malformation of the inner ear, specifically the cochlea. It is associated with sensorineural hearing loss and may also be associated with recurrent meningitis.

Both these conditions are typically diagnosed via imaging studies like CT or MRI scans of the temporal bone. Management often involves cochlear implants, but the outcomes can vary depending on the severity of the malformation and associated complications.

-----X-----X-----

Q: Hearing loss causes and audiological test

Causes of Hearing Loss

Category	Specific Causes
Genetic Factors	Congenital anomalies (e.g., Pendred syndrome, Usher syndrome) Genetic mutations affecting ear structure or function
Infections	Congenital infections (e.g., CMV, rubella)

	Acquired infections (e.g., meningitis, measles, mumps)
Anatomical Abnormalities	Malformations of the ear canal or middle ear Otosclerosis
Trauma	Head injuries Acoustic trauma (e.g., exposure to loud noise) Barotrauma
Ototoxicity	Medications (e.g., aminoglycosides, cisplatin) Chemicals (e.g., solvents, heavy metals)
Age-Related Changes	Presbycusis (age-related hearing loss)
Other Causes	Tumors (e.g., acoustic neuroma) Autoimmune diseases Meniere's disease Sudden idiopathic hearing loss

Audiological Tests

Test	Description	Purpose
Pure Tone Audiometry	Measures hearing sensitivity across a range of frequencies	Determines the degree and type of hearing loss (conductive, sensorineural, or mixed)
Tympanometry	Assesses the function of the middle ear	Identifies middle ear pathologies (e.g., fluid, otosclerosis, eustachian tube dysfunction)
Speech Audiometry	Evaluates understanding and clarity of speech	Assesses speech recognition and comprehension abilities
Otoacoustic Emissions (OAEs)	Measures sounds generated by the cochlea	Screens for cochlear (inner ear) function, especially in newborns and infants
Auditory Brainstem Response (ABR)	Records brainwave activity in response to sound	Assesses the integrity of the auditory pathway up to the brainstem
Electrocochleography (ECoG)	Measures electrical potentials generated in the inner ear	Useful in diagnosing Meniere's disease and other inner ear disorders
Vestibular Evoked Myogenic Potentials (VEMP)	Assesses the saccule and the inferior vestibular nerve	Helps in diagnosing vestibular disorders associated with hearing loss
Immittance Audiometry	Measures the impedance of the middle ear	Helps in identifying middle ear disorders

-----X-----X-----

Q: Hearing Assessment Screening in Children

Early detection and intervention for hearing loss in children are vital for optimal speech and developmental outcomes.

Regular screening, coupled with appropriate follow-up and management, can significantly impact a child's communication abilities and overall quality of life.

- **Purpose:** Early identification of hearing loss to facilitate timely intervention and support optimal language, cognitive, and social development.
- **Age Groups:** Newborns, infants, toddlers, and school-aged children.

Screening Methods

1. Newborn Hearing Screening

- **Otoacoustic Emissions (OAE):** Measures sound waves produced in the inner ear. Absence of these sounds can indicate hearing loss.
- **Automated Auditory Brainstem Response (AABR):** Assesses the auditory nerve and brainstem's response to sound.

2. Infants and Toddlers

- **Behavioral Audiometry:** Observes child's response to sounds (e.g., turning head towards sound).
- **Visual Reinforcement Audiometry (VRA):** Child is trained to look towards a sound source, often paired with a visual reward.

3. Older Children

- **Play Audiometry:** Child is engaged in a play activity (e.g., placing a block in a box) in response to hearing a sound.
- **Pure-Tone Audiometry:** Child wears headphones and responds to tones at different pitches and volumes.

Indications for Screening

- **Universal Newborn Hearing Screening (UNHS):** Recommended for all newborns.
- **High-Risk Factors:** Family history of hearing loss, neonatal intensive care unit (NICU) stay >5 days, exposure to ototoxic medications, syndromes associated with hearing loss, postnatal infections, head trauma, and recurrent otitis media.

- **Developmental Delays:** Speech or language delay, lack of response to sound, or parental concerns about hearing.

Follow-Up and Diagnosis

- **Referral for Diagnostic Testing:** If screening results indicate potential hearing loss.
- **Diagnostic Audiological Evaluation:** Comprehensive testing to confirm and quantify the degree of hearing loss.
- **Genetic Testing and Imaging:** In some cases, to identify etiology.

Management

- **Early Intervention Services:** Crucial for children with confirmed hearing loss.
- **Hearing Aids or Cochlear Implants:** Depending on the degree and type of hearing loss.
- **Speech and Language Therapy:** To support communication development.
- **Regular Monitoring:** To assess hearing status and developmental progress.

Challenges and Considerations

- **False Positives/Negatives:** Possible in screening tests, necessitating careful follow-up.
- **Access and Compliance:** Ensuring all children, especially in underserved areas, receive screening and necessary follow-up.
- **Parental Education and Support:** Critical for ongoing management and intervention.

Public Health Implications

- **Policy and Advocacy:** Support for universal screening programs and access to intervention services.

-----X-----X-----

Q: Describe the etiopathogenesis and clinical features in a 3 year old child with hearing impairment. What are the laboratory tests for assessment of such a child?

Etiopathogenesis of Hearing Impairment in a 3-Year-Old Child

- **Genetic Factors:** Family history of hearing loss, especially if autosomal recessive or dominant.

- **Perinatal Factors:** Prematurity, low birth weight, neonatal jaundice, or anoxia.
- **Infections:** Congenital infections like CMV, or acquired ones like meningitis or recurrent otitis media.
- **Trauma:** Head injuries affecting the auditory pathway.
- **Ototoxic Drugs:** Exposure to medications like aminoglycosides.
- **Environmental Factors:** Exposure to loud noise, especially if chronic.

Clinical Features

- **Delayed Speech Development:** One of the earliest signs, especially if the child is not making age-appropriate sounds or words.
- **Inattentiveness:** May appear to be ignoring when called or spoken to.
- **Volume Control:** Watching TV or listening to music at a high volume.
- **Frequent Ear Infections:** May have a history of recurrent otitis media.
- **Behavioral Issues:** Frustration or tantrums, possibly due to inability to communicate or understand.
- **Poor Academic Performance:** Difficulty in understanding and following instructions can affect learning.

Laboratory Tests for Assessment

- **Audiometry:** To assess the degree and type of hearing loss.
- **Tympanometry:** To evaluate middle ear function.
- **Otoacoustic Emissions (OAE):** To check the inner ear's response to sound.
- **Auditory Brainstem Response (ABR):** To evaluate the auditory nerve and pathway.
- **CT/MRI:** To rule out structural abnormalities like Michel's or Mondini's aplasia.
- **Genetic Testing:** If a hereditary component is suspected.
- **Blood Tests:** To check for infections or conditions like hypothyroidism that could contribute to hearing loss.

Management may include hearing aids, cochlear implants, and speech therapy, depending on the type and degree of hearing loss.

-----X-----X-----

Q: Universal Neonatal Hearing Screening (UNHS)

Rationale

- **Early Identification:** Detect hearing loss as early as possible to initiate timely intervention.
- **Critical Period:** The first few months of life are crucial for auditory pathway and language development.
- **High Incidence:** Hearing loss is one of the most common congenital anomalies.

Screening Methods

- **Otoacoustic Emissions (OAE):** Measures sound waves produced in the inner ear. Quick and non-invasive.
- **Automated Auditory Brainstem Response (AABR):** Measures how the hearing nerve responds to sound. More comprehensive but also more time-consuming.

Timing

- **Initial Screening:** Within the first 48 hours after birth or before hospital discharge.
- **Follow-up Screening:** If initial screening is failed, a repeat test is done within the first month.
- **Diagnostic Evaluation:** If the follow-up screening is also failed, a full audiological evaluation is recommended within the first three months.

Challenges

- **Cost:** High initial setup and running costs.
- **Manpower:** Requires trained personnel for screening and follow-up.
- **Parental Anxiety:** False positives can cause unnecessary stress.

Benefits

- **Early Intervention:** Allows for immediate action, such as hearing aids or cochlear implants, and enrollment in early intervention services.
- **Better Outcomes:** Improved language, social, and academic development.

Current Guidelines

- **Joint Committee on Infant Hearing (JCIH):** Recommends universal screening of all newborns.

- **American Academy of Pediatrics:** Endorses JCIH recommendations and suggests that all newborns should be screened, regardless of risk factors.

Universal Neonatal Hearing Screening is considered the standard of care in many developed countries and is increasingly being adopted in developing nations as well.

-----X-----X-----

Q: Pediatric Cochlear Implant

Indications

- **Profound Sensorineural Hearing Loss:** Bilateral, unaided.
- **Limited Benefit from Hearing Aids:** Lack of progress in the development of auditory skills.
- **Age:** As young as 12 months for profound hearing loss.

Preoperative Assessment

- **Audiological Evaluation:** Confirm the type and degree of hearing loss.
- **Medical Evaluation:** Rule out contraindications like active middle ear infection.
- **Speech and Language Assessment:** Evaluate current communication skills.
- **Psychological Assessment:** Assess family readiness and support.

Surgical Procedure

- **Mastoidectomy and Tympanotomy:** Access to the middle ear.
- **Electrode Insertion:** Into the cochlea.
- **Device Fixation:** Implant is secured and tested.

Postoperative Care

- **Device Activation:** Usually 2-4 weeks post-surgery.
- **Mapping:** Customizing the sound processor settings.
- **Regular Follow-ups:** For device adjustments and monitoring.

Rehabilitation

- **Auditory-Verbal Therapy:** To maximize hearing and speech capabilities.
- **Speech Therapy:** For language development.
- **Family Involvement:** Crucial for successful outcomes.

Risks and Complications

- **Surgical Risks:** Infection, facial nerve damage, meningitis.
- **Device Failure:** Rare but possible.
- **Psychological:** Unrealistic expectations, adjustment issues.

Outcomes

- **Speech Recognition:** Significant improvement in most cases.
- **Language Development:** Catches up to age-appropriate levels in many cases.
- **Quality of Life:** Generally improved social interaction and academic performance.

Cost-Effectiveness

- **High Initial Cost:** But considered cost-effective in the long term due to improved quality of life and decreased societal costs.

Cochlear implants have revolutionized the management of severe-to-profound hearing loss in children, offering the opportunity for near-normal auditory experience and spoken language development.

-----X-----

Q: Acute Suppurative Otitis Media (ASOM) in Children; Outline the etiopathogenesis of acute suppurative otitis media. Discuss in brief the treatment and complications of acute suppurative otitis media (ASOM) in children.

Etiopathogenesis

- **Predisposing Factors:** Upper respiratory tract infections, eustachian tube dysfunction, exposure to smoke, daycare attendance.
- **Microbial Agents:** Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis.
- **Pathogenesis:** Infection of the middle ear space leading to pus formation, causing tympanic membrane bulging and pain. On examination, TM will be dull and congested, pus point may be visible. Pain relieves on spontaneous rupture with flow of pus along EAC

Treatment

Medical Management

Lab:- Elevated inflammatory markers, leucocytosis; Pus culture to isolate pathogen

- **Antibiotics:**
 - Amoxicillin (80-90 mg/kg/day) for 7-10 days is the first-line treatment. Later guided by sensitivity pattern.
 - In penicillin-allergic patients, alternatives include cefdinir, cefuroxime, or Erythromycin/azithromycin.
- **Analgesics:**
 - Paracetamol or ibuprofen for pain relief.
- **Decongestants:**
 - Not recommended for children under 6 years.
- Antihistaminics like levocetirizine
- **Steroids:**
 - Controversial; may be considered in severe cases after achieving infection control.

Surgical Management

- **Myringotomy:** Incision in the tympanic membrane to relieve pressure.
- **Tympanostomy Tubes:** In recurrent cases or treatment failure.

Follow-up

- **Re-examination:** 2-4 weeks post-treatment to confirm resolution.
- **Audiological Evaluation:** If symptoms persist or in recurrent cases.

Complications

Local Complications

- **Chronic Suppurative Otitis Media:** Persistent infection.
- **Tympanic Membrane Perforation:** May lead to hearing loss.
- **Cholesteatoma:** Abnormal skin growth in the middle ear.

Systemic Complications

- **Mastoiditis:** Infection of the mastoid bone.
- **Facial Nerve Paralysis:** Rare but serious.

- **Intracranial Complications:** Meningitis, brain abscess, lateral sinus thrombosis.

Prevention

- **Vaccination:** Pneumococcal and influenza vaccines.
- **Breastfeeding:** Encouraged for the first 6 months. Avoid bottle feeding
- **Avoid Smoke Exposure:** Passive smoking is a risk factor. Avoid cooking with wood/tandoor; keep children exposure to minimum possible duration to these, Usage of LPG and well ventilated household

-----X-----X-----

Q: Recurrent Acute Otitis Media (RAOM) in Children

Definition

- **RAOM:** Three or more episodes of acute otitis media (AOM) within a 6-month period or four or more episodes in a year.

Etiopathogenesis

- **Predisposing Factors:** Young age, daycare attendance, bottle-feeding, family history, upper respiratory infections, allergies.
- **Microbial Agents:** (Similar to AOM) - Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis.

Diagnosis

- **Clinical Evaluation:** History of recurrent ear infections, otoscopic findings.
- **Audiological Tests:** To assess hearing loss.
- **Tympanometry:** To evaluate middle ear function.

Treatment

Medical Management

- **Prophylactic Antibiotics:**
 - Controversial; may include low-dose amoxicillin.
- **Immunotherapy:**
 - Anti-pneumococcal vaccine. PPV -23 for 2yrs and above
- **Topical Nasal Steroids:**

- For children with allergic rhinitis.

Surgical Management

- **Tympanostomy Tubes:**
 - Indicated for persistent effusion and hearing loss.
- **Adenoidectomy:**
 - Considered in children over 4 years with nasal obstruction.

Complications

- **Hearing Loss:** Temporary or permanent.
- **Speech Delay:** Due to hearing impairment.
- **Chronic Otitis Media:** With or without effusion.

Prevention

- **Vaccination:** Pneumococcal and influenza vaccines.
- **Breastfeeding:** For at least the first 6 months.
- **Hygiene Measures:** Handwashing, avoiding bottle propping.

Current Guidelines

- **American Academy of Otolaryngology:** Recommends against routine antibiotic prophylaxis; advocates for watchful waiting and symptom relief. No practice guidelines from Indian Bodies

Follow-up

- **Regular Monitoring:** To assess treatment efficacy and detect complications.
- **Audiological Assessments:** Periodic hearing tests.

-----X-----X-----

Q: Investigations and Treatment of Childhood Vertigo

Investigations

- **Clinical History and Physical Examination:** To rule out systemic/ cerebellar causes and assess for nystagmus.
- **Audiological Tests:** To evaluate hearing function.
- **Videonystagmography (VNG):** To assess eye movements and balance.

- **MRI or CT Scan:** To rule out structural abnormalities or tumors.
- **Electroencephalogram (EEG):** To rule out seizures.
- **Blood Tests:** To check for metabolic or infectious causes.

Treatment

Medical Management

- **Antihistamines:**
 - Meclizine, cinnarizine, dimenhydrinate for symptomatic relief.
- **Anti-emetics:**
 - Ondansetron for nausea and vomiting.
- **Benzodiazepines:**
 - Diazepam; used cautiously due to sedative effects.
- **Anticholinergics:**
 - Scopolamine patches; limited use in children.
- **Steroids:**
 - For inflammatory causes; e.g., dexamethasone.

Behavioral Therapy

- **Vestibular Rehabilitation:** Exercises to improve balance and spatial orientation.

Surgical Management

- **Endolymphatic Sac Decompression:** For Meniere's disease.
- **Vestibular Nerve Section:** Rare; for severe, unresponsive cases.

Special Considerations

- **Migraine Prophylaxis:**
 - Propranolol or Topiramate for vestibular migraines.
- **Dietary Modifications:**
 - Low salt diet in Meniere's disease.

Follow-up and Monitoring

- **Regular Clinical Assessments:** To evaluate treatment efficacy.
- **Audiological Monitoring:** To assess hearing and balance.

Complications

- **Persistent Symptoms:** Failure to treat can lead to chronic symptoms.
- **Hearing Loss:** In untreated Meniere's disease or vestibular neuritis.

-----X-----X-----



SKIN

Q: Clinical conditions associated with Maculopapular rashes in children and their differential diagnosis.

Clinical Condition	Differential Diagnosis	Key Features of Condition
Measles	Rubella, Roseola, Scarlet Fever	High fever, cough, coryza, Koplik spots, rash starts from head and spreads downward
Rubella (German Measles)	Measles, Scarlet Fever, Fifth Disease	Low-grade fever, lymphadenopathy, rash starts on face and spreads downward
Roseola (Sixth Disease)	Measles, Rubella, Scarlet Fever	High fever followed by rash, mostly affects infants and toddlers
Scarlet Fever	Measles, Rubella, Kawasaki Disease	High fever, sore throat, "strawberry tongue," sandpaper-like rash
Fifth Disease (Erythema Infectiosum)	Rubella, Measles, Scarlet Fever	"Slapped cheek" appearance, lacy rash on extremities
Chickenpox (Varicella)	Smallpox, Impetigo, Herpes Simplex	Itchy rash with vesicles, starts centrally and spreads outward
Drug Eruption	Viral Exanthem, Contact Dermatitis	History of drug exposure, may have systemic symptoms
Kawasaki Disease	Scarlet Fever, Juvenile Rheumatoid Arthritis, Toxic Shock Syndrome	High fever, conjunctivitis, "strawberry tongue," rash, and peeling of the skin especially on hands and feet
Dengue Fever	Zika, Chikungunya, Yellow Fever	High fever, severe headache, joint and muscle pain, rash may appear in severe cases
Hand, Foot, and Mouth Disease	Impetigo, Chickenpox	Sores in mouth and a rash on hands and feet

Infectious Mononucleosis	CMV, Toxoplasmosis, Acute HIV	Fever, sore throat, swollen lymph nodes, rash may occur especially if treated with ampicillin or amoxicillin
---------------------------------	-------------------------------	--

-----X-----X-----

Q: Diagnosis and management of a 2-year-old child with Petechial skin rash

Diagnosis of a 2-year-old child with Petechial Skin Rash

Clinical Evaluation

- Detailed history: Onset, duration, associated symptoms like fever, joint pain, etc.
- Physical examination: To assess the extent and distribution of the rash, and to look for other signs like fever, lymphadenopathy, etc.

Laboratory Tests

- Complete Blood Count (CBC): To assess platelet count and look for signs of infection or anemia.
- Coagulation profile: PT, aPTT, INR to assess clotting.
- Blood cultures: To rule out bacterial sepsis.
- Liver and renal function tests: To rule out systemic causes.
- Lumbar puncture: If meningococcal infection is suspected.

Imaging

- Chest X-ray: If respiratory symptoms are present.
- Ultrasound: To assess spleen and liver size if needed.

Management of a 2-year-old child with Petechial Skin Rash

General Measures

- Hospitalization: May be required for severe cases or if sepsis is suspected.
- Hydration: Adequate fluid intake.

Pharmacotherapy

- Antibiotics: Empirical antibiotics like Ceftriaxone or Penicillin if bacterial infection is suspected, pending culture results.

- Dose: Ceftriaxone 50-75 mg/kg/day IV in divided doses
- Common Side Effects: Diarrhea, rash, elevated liver enzymes
- Antihistamines: For symptomatic relief from itching if present.
 - Dose: Diphenhydramine 1-2 mg/kg/dose, up to 6 doses per day
 - Common Side Effects: Drowsiness, dry mouth
- Corticosteroids: In cases of immune-mediated causes.
 - Dose: Prednisolone 1-2 mg/kg/day
 - Common Side Effects: Weight gain, mood changes

Monitoring

- Regular CBC to monitor platelet count.
- Vital signs monitoring.
- Blood cultures to tailor antibiotic therapy.

Differential Diagnosis of a 2-year-old child with Petechial Skin Rash

Condition	Clinical Features	Diagnostic Tests	Treatment Approach
Meningococcal Sepsis	High fever, altered mental status, purpura, shock	Blood culture, lumbar puncture	IV antibiotics, supportive care
Henoch-Schönlein Purpura	Abdominal pain, joint pain, renal involvement, palpable purpura	Urinalysis, CBC	Supportive care, steroids if severe
ITP (Idiopathic Thrombocytopenic Purpura)	Isolated petechiae, easy bruising, otherwise well	CBC, peripheral smear	Observation, IVIG or steroids if severe

Leukemia	Fatigue, pallor, petechiae, easy bruising, bone pain	CBC, bone marrow biopsy	Chemotherapy, supportive care
Viral Infections	Fever, URI symptoms, petechial rash	Viral PCR, serology	Supportive care
Rocky Mountain Spotted Fever	Fever, headache, abdominal pain, petechial rash starting on extremities	Serology, PCR	Doxycycline
Dengue Fever	High fever, severe headache, joint and muscle pain, petechial rash	Dengue serology, PCR	Supportive care, fluids
Scarlet Fever	Fever, sore throat, "strawberry tongue," fine sandpaper-like rash, petechiae	Throat culture	Antibiotics (Penicillin)
Eczema (Dermatitis)	Itching, redness, sometimes petechiae from scratching	Clinical diagnosis	Topical steroids, antihistamines
Drug Reaction	Variable fever, rash may be urticarial, maculopapular, or petechial	Drug history, skin testing	Discontinue drug, antihistamines

-----X-----X-----

Q: Approach to a Case of Exanthematous Fever in a 14-Month-Old Child**Initial Assessment**

- **Vital Signs:** Check temperature, heart rate, respiratory rate, and blood pressure.
- **General Examination:** Assess hydration status, look for lymphadenopathy, and examine the oral cavity.

History Taking

- **Onset and Duration:** When did the fever and rash start?
- **Associated Symptoms:** Cough, runny nose, sore throat, vomiting, diarrhea, etc.
- **Exposure History:** Contact with sick individuals, travel history, etc.
- **Immunization Status:** Particularly MMR (Measles, Mumps, Rubella) vaccine.

Physical Examination

- **Skin:** Characterize the rash (maculopapular, vesicular, etc.), and note its distribution.
- **Respiratory, Cardiovascular, Abdominal Examination:** To rule out complications or other associated conditions.

Diagnostic Tests

- **Complete Blood Count (CBC):** To assess for infection or anemia.
- **Liver Function Tests (LFTs):** If indicated.
- **Throat Swab and Culture:** If pharyngitis is present.
- **Urine Analysis:** To rule out UTI which can sometimes present with fever and rash.
- **Blood Culture:** If bacterial infection is suspected.
- **Serological Tests:** Such as IgM ELISA for specific viral etiologies (e.g., Measles, Rubella).

Management**Pharmacotherapy**

- **Antipyretics:** Paracetamol for fever control.
 - Dose: 10-15 mg/kg/dose every 4-6 hours as needed
 - Common Side Effects: Liver toxicity in overdose
- **Antihistamines:** For symptomatic relief from itching.

- Dose: Diphenhydramine 1-2 mg/kg/dose, up to 6 doses per day
- Common Side Effects: Drowsiness, dry mouth
- **Antibiotics:** Only if bacterial superinfection is suspected.
 - Dose: Amoxicillin 20-40 mg/kg/day in divided doses
 - Common Side Effects: Diarrhea, rash

Supportive Care

- **Hydration:** Oral rehydration solution or IV fluids if needed.
- **Isolation:** To prevent the spread of infection, especially if viral etiology is suspected.

Monitoring and Follow-up

- **Regular Monitoring:** Of vital signs and hydration status.
- **Follow-up:** To assess the resolution of symptoms and any complications.

Referral Criteria

- **Non-resolving Fever:** Fever persisting beyond 5 days.
- **Complications:** Such as respiratory distress, severe dehydration, or neurologic symptoms.

Differential Diagnosis for Exanthematous Fever in a 14-Month-Old Child

Condition	Clinical Features	Diagnostic Tests	Treatment Approach
Measles	High fever, cough, coryza, conjunctivitis, Koplik spots, maculopapular rash	IgM ELISA, viral culture	Supportive care, Vitamin A
Rubella	Low-grade fever, mild rash, lymphadenopathy	IgM ELISA	Supportive care
Roseola (HHV-6)	High fever followed by rash as fever resolves	Clinical diagnosis	Supportive care
Scarlet Fever	Fever, sore throat, "strawberry tongue," fine sandpaper-like rash	Throat culture	Antibiotics (Penicillin)

Chickenpox (Varicella)	Fever, vesicular rash	Tzanck smear, PCR	Supportive care, antivirals if severe
Hand, Foot, and Mouth	Low-grade fever, vesicular rash on hands, feet, and oral mucosa	Clinical diagnosis	Supportive care
Erythema Infectiosum	"Slapped cheek" rash, lacy rash on extremities	Parvovirus B19 IgM	Supportive care
Kawasaki Disease	Persistent fever, conjunctivitis, rash, mucosal changes, extremity changes	Echocardiogram, CRP, ESR	IVIG, Aspirin
Drug Reaction	Variable fever, rash may be urticarial, maculopapular, or petechial	Drug history, skin testing	Discontinue drug, antihistamines
Bacterial Sepsis	High fever, may have petechial or purpuric rash	Blood culture, CBC, CRP	IV antibiotics, supportive care

-----X-----X-----

Q: Differential Diagnosis for Bullous Skin Eruptions in Newborn Babies

Condition	Clinical Features	Diagnostic Tests	Treatment Approach
Bullous Impetigo	Superficial, easily ruptured bullae filled with yellow fluid; often near diaper area	Culture of fluid	Topical or systemic antibiotics
Neonatal Herpes Simplex	Vesicular and bullous lesions, may have systemic involvement like fever, lethargy	PCR, viral culture	IV Acyclovir
Epidermolysis Bullosa	Fragile skin, blisters from minor trauma, may involve mucous membranes	Skin biopsy	Wound care, pain management
Congenital Pemphigus	Flaccid bullae, often ruptured, leaving erosions; may involve oral mucosa	Skin biopsy, immunofluorescence	Steroids, immunosuppressants

Staphylococcal Scalded Skin Syndrome	Diffuse erythema, tender skin, and bullae leading to skin desquamation	Culture, biopsy	IV antibiotics, wound care
Bullous Mastocytosis	Urticaria pigmentosa-like lesions, bullae may form; may have systemic symptoms	Serum tryptase, skin biopsy	Antihistamines, mast cell stabilizers
Incontinentia Pigmenti	Erythematous, vesicular rash at birth progressing to hyperpigmented streaks	Skin biopsy, genetic testing	Supportive care, treat complications
Bullous Congenital Ichthyosiform Erythroderma	Large, dark, plate-like scales covering the body, may have bullae	Skin biopsy	Emollients, retinoids
Transient Bullous Dermolysis of the Newborn	Bullae over erythematous base, usually resolves spontaneously	Clinical diagnosis	Supportive care
Linear IgA Bullous Dermatitis	Tense bullae on an erythematous base, may have "cluster of jewels" appearance	Direct immunofluorescence	Dapsone, steroids

-----X-----X-----

Q: Enumerate the causes of persistent fever which are not due to infection.

Describe the clinical presentation of ectodermal dysplasias.

Causes of Persistent Fever Not Due to Infection (Non infectious causes of fever)

1. Autoimmune Diseases
 - Rheumatoid Arthritis
 - Systemic Lupus Erythematosus
 - Juvenile Idiopathic Arthritis
2. Inflammatory Conditions

- Inflammatory Bowel Disease
 - Vasculitis
 - Sarcoidosis
3. Malignancies
- Leukemia
 - Lymphoma
 - Neuroblastoma
4. Drug-Induced Fever
- Antibiotics
 - Antipsychotics
 - Chemotherapy agents
5. Endocrine Disorders
- Hyperthyroidism
 - Adrenal insufficiency
 - Pheochromocytoma
6. Hematologic Disorders
- Hemolytic Anemia
 - Thrombocytopenia
7. Genetic Syndromes
- Familial Mediterranean Fever
 - Hyper-IgD Syndrome
8. Miscellaneous
- Factitious Fever
 - Teething in infants (controversial)

Clinical Presentation of Ectodermal Dysplasias

Ectodermal dysplasias are a heterogeneous group of disorders, and not all features may be present in every individual. Diagnosis involves genetic testing and multidisciplinary evaluation.

Feature	Description
Skin	Dry, scaly skin; eczema; hyperpigmentation or hypopigmentation
Hair	Sparse, fine, or absent hair on the scalp, eyebrows, and eyelashes
Nails	Dystrophic, ridged, or absent nails
Teeth	Hypodontia or anodontia; conical or peg-shaped teeth
Sweat Glands	Reduced or absent, leading to heat intolerance
Mucous Membranes	Dryness in the eyes, mouth, and airways; may lead to respiratory issues
Olfactory Sense	Reduced or absent
Auditory Features	Hearing loss due to malformation or absence of middle ear structures
Ocular Features	Dry eyes, cataracts, astigmatism
Temperature Regulation	Difficulty due to lack of sweat glands
Feeding Difficulties	May occur in infancy due to cleft lip/palate or oral dryness
Other Features	Frequent respiratory infections, skin infections due to poor barrier function

-----X-----X-----

Q: Scabies in Children

Etiology

- Caused by the mite *Sarcoptes scabiei* var. *hominis*
- Transmitted through close personal contact or contaminated objects

Clinical Presentation

Feature	Description
Itching	Intense pruritus, especially at night

Skin Lesions	Erythematous papules, vesicles, and burrows
Distribution	Commonly affects interdigital spaces, wrists, axillae, abdomen, and genitalia
Secondary Infections	Impetigo, cellulitis due to bacterial superinfection
Family History	Often other family members are affected

Diagnosis

- Clinical examination
- Dermoscopy to visualize mites, eggs, or feces
- Skin scraping and microscopic examination
- Occasionally, PCR for mite DNA

Management

Treatment	Dose and Duration	Notes
Permethrin Cream	5%, apply to all skin from neck down, overnight for 8-14 hours	First-line treatment; repeat after 7 days
Ivermectin	200 µg/kg orally, repeat in 7 days	Used in severe cases or treatment failure
Lindane	Not recommended in children	Neurotoxic
Crotamiton	Apply daily for 5 days	Less effective, used in mild cases
Antihistamines	To control itching	E.g., cetirizine, hydroxyzine
Topical Steroids	For eczematous changes	Short-term use
Antibiotics	If secondary bacterial infection	E.g., topical or oral antibiotics like mupirocin or cephalexin

Prevention

- Treat all close contacts
- Wash all bedding, clothing, and towels in hot water
- Vacuum home and discard the vacuum bag

Complications

- Secondary bacterial infections

- Post-scabies eczema
- Psychological distress due to itching

Norwegian Scabies (Crusted Scabies)

Clinical Presentation

- Hyperkeratotic, crusted plaques
- **Affects immunocompromised individuals**
- May be pruritic or asymptomatic
- Commonly affects palms, soles, scalp, and trunk

Diagnostic Approach

- Clinical examination
- Skin scraping for microscopic examination
- Dermoscopy

Topical Keratolytics (e.g., salicylic acid) to remove crusts; enhances penetration of scabicides are used in addition to aggressive anti scabies therapy.

-----X-----X-----

Q: Contact dermatitis

- ❖ Contact dermatitis is a multifaceted dermatological condition requiring a nuanced approach to diagnosis and management
- ❖ Understanding its complex pathophysiology aids in targeted therapy
- ❖ The management strategy should be individualized, considering the patient's lifestyle, occupational exposures, and the severity of the condition
- ❖ Ongoing research and emerging therapies continue to evolve the landscape of treatment options, offering hope for more effective management in challenging cases.

Pathophysiology

1. **Irritant Contact Dermatitis (ICD):**

- Direct cytotoxic effect on keratinocytes.

- Activation of innate immunity.
- Non-immunologic mechanism; no prior sensitization required.

2. **Allergic Contact Dermatitis (ACD):**

- Type IV hypersensitivity reaction.
- Involves sensitization phase and elicitation phase.
- Langerhans cells capture allergen, migrate to lymph nodes, present to T-cells.

Clinical Presentation

- **Acute Phase:**
 - Erythema, edema, vesicles.
 - Pruritus is a predominant symptom.
- **Chronic Phase:**
 - Lichenification, scaling, fissuring.
 - Hyperpigmentation or hypopigmentation in darker skin types.

Differential Diagnosis

- Atopic dermatitis, psoriasis, drug eruptions, photoallergic dermatitis.

Diagnostic Approach

- **Detailed History:** Occupational, hobbies, recent exposures, personal/family history of atopy.
- **Physical Examination:** Distribution pattern, morphology of lesions.
- **Patch Testing:** Gold standard for ACD; identifies specific allergens.
- **Other Tests:** Skin biopsy in atypical cases.

Advanced Management

1. **Topical Therapeutics:**
 - Potent topical corticosteroids: First-line for acute inflammation.
 - Calcineurin inhibitors: For sensitive areas like face, intertriginous areas.
 - Barrier creams and emollients: Restore skin barrier function.
2. **Systemic Therapies:**

- Oral corticosteroids: Short courses for severe acute flares.
- Cyclosporine, methotrexate, azathioprine: Considered in chronic, severe, refractory cases.

3. **Phototherapy:**

- Narrowband UVB or PUVA therapy for chronic, extensive disease.

4. **Emerging Therapies:**

- Biologics targeting specific cytokines involved in inflammation.
- Janus kinase (JAK) inhibitors.

Patient Education

- Importance of avoiding identified allergens.
- Proper use of personal protective equipment.
- Skin care regimen to maintain barrier integrity.

Occupational Considerations

- Identification and substitution of allergens in the workplace.
- Education about protective measures.

Research and Future Directions

- Exploration of the role of skin microbiome in contact dermatitis.
- Development of novel topical and systemic therapeutics.
- Genetic predisposition and its implications in disease manifestation.

-----X-----X-----

Q: Management of Vascular Nevi in Children

Types of Vascular Nevi

- Hemangiomas
- Port-wine stains
- Venous malformations
- Lymphatic malformations

- Arteriovenous malformations

Diagnostic Approach

- Clinical examination
- Dermoscopy
- Imaging studies: MRI, Doppler ultrasound
- Biopsy for uncertain cases

Management Strategies

Type of Vascular Nevi	Treatment Options	Notes
<i>Hemangiomas</i>	Observation Oral propranolol Topical timolol Steroid injections	Most regress spontaneously; propranolol is first-line for problematic cases
<i>Port-wine stains</i>	Pulsed dye laser Topical rapamycin	Multiple sessions may be needed; early treatment preferred
<i>Venous malformations</i>	Sclerotherapy Surgical excision	Choice depends on location and extent
<i>Lymphatic malformations</i>	Sclerotherapy Surgical excision	Complex cases may require multi-disciplinary approach
<i>Arteriovenous malformations</i>	Embolization Surgical excision	High risk of bleeding; consult interventional radiology

Follow-up and Monitoring

- Regular dermatological assessment
- Monitor for complications such as ulceration, infection, or functional impairment
- Psychological support for cosmetic concerns

Complications

- Bleeding
- Ulceration
- Infection

- Functional impairment
- Cosmetic disfigurement

-----X-----X-----

Q: Stevens-Johnson Syndrome (SJS)

Etiology

- **Drug-Induced:** Most commonly implicated drugs include sulfonamides, antiepileptic drugs like phenytoin and carbamazepine, allopurinol, and nonsteroidal anti-inflammatory drugs (NSAIDs).
- **Infectious Agents:** Mycoplasma pneumoniae, viral infections like herpes simplex and Epstein-Barr virus.
- **Genetic Factors:** Certain HLA types (e.g., HLA-B*1502) are associated with a higher risk of SJS, particularly in response to specific medications like carbamazepine.
- **Idiopathic:** In a significant number of cases, the etiology remains unknown despite extensive investigation.

Clinical Features

- **Prodromal Phase:** Fever, malaise, and upper respiratory symptoms may precede the skin manifestations by 1-3 days.
- **Cutaneous Manifestations:** Begins with erythematous or purpuric macules, rapidly progressing to epidermal detachment and formation of flaccid bullae.
- **Mucosal Involvement:** Painful erosions and ulcerations involving the oral, ocular, and genital mucosa.
- **Systemic Symptoms:** High fever, tachycardia, and general malaise.

Diagnostic Criteria

- **Clinical Presentation:** The diagnosis is primarily clinical, based on the characteristic skin and mucosal lesions.
- **Skin Biopsy:** Epidermal necrosis and full-thickness epidermal detachment.
- **Blood Tests:** Elevated inflammatory markers, liver function tests, and complete blood count to assess the severity and rule out other causes.
- **Ophthalmological Examination:** To assess the extent of ocular involvement.

Management

- **Immediate Cessation of Offending Agent:** The first and foremost step is to discontinue the suspected causative drug immediately.
- **Hospitalization:** Preferably in a burn unit or ICU for close monitoring.
- **Supportive Care:**
 - Fluid resuscitation
 - Electrolyte management
 - Nutritional support
- **Wound Management:**
 - Sterile non-adherent dressings
 - Topical antiseptics
- **Systemic Treatment:**
 - Corticosteroids: Controversial and generally not recommended due to lack of proven benefit and potential for complications.
 - Intravenous Immunoglobulin (IVIG): Used in severe, life-threatening cases.
 - Cyclosporine: Some evidence for effectiveness in severe cases.
- **Antibiotics:** Only if secondary bacterial infection is suspected or confirmed.
- **Ocular Care:** Lubricating eye drops, topical steroids, and consultation with an ophthalmologist.
- **Pain Management:** Topical anesthetics for mucosal lesions and systemic analgesics.

Treatment Options	Notes/Comments
Discontinue Offending Agent	Immediate cessation of suspected drug
Supportive Care	Fluid resuscitation, electrolyte balance, nutritional support

Wound Care	Sterile dressings for skin lesions, eye care for ocular involvement
Systemic Corticosteroids	Controversial; may be considered in early stages
Intravenous Immunoglobulin (IVIG)	Used in severe cases; dose varies
Antibiotics	For secondary bacterial infections
Pain Management	Topical anesthetics for mucosal lesions, systemic analgesics as needed

Complications

- **Secondary Infections:** Risk of bacterial superinfection of skin lesions.
- **Ocular Complications:** Corneal ulcers, conjunctivitis, potential for blindness.
- **Long-term Sequelae:** Scarring, changes in skin pigmentation, chronic ocular complications, and systemic organ involvement.
- **Psychological Impact:** The condition can have a long-term psychological impact, requiring mental health support.

Prognosis

- **Mortality:** Ranges from 5% to 15%, higher in elderly and those with extensive skin involvement.
- **Long-term Outcome:** Depends on the promptness of diagnosis and management, as well as the extent of skin and organ involvement.

Follow-Up

- **Dermatological:** To assess healing and scarring.
- **Ophthalmological:** To monitor and manage ocular complications.
- **Psychological:** Support for the mental health impact of the disease.

-----X-----X-----

Q: Erythema Multiforme

Characteristic Clinical Features

- **Prodromal Symptoms:** Fever, malaise, and upper respiratory symptoms may precede skin lesions.
- **Cutaneous Lesions:**
 - **Target lesions** are the hallmark, consisting of three zones: a central dusky area, a pale edematous ring, and a peripheral erythematous halo.
 - Lesions may be symmetrically distributed, often affecting the extremities.
- **Mucosal Involvement:** Oral, genital, and ocular mucosa may be affected, but less severely than in Stevens-Johnson Syndrome.
- **Pruritus or Pain:** Lesions may be pruritic or painful but are generally less severe compared to other similar dermatological conditions.
- **Systemic Symptoms:** Generally mild and may include low-grade fever and malaise.
- **Associations:** associated with inflammatory bowel disease, malignancies, and infections- HSV, Epstein-Barr virus, cytomegalovirus, hepatitis C, and influenza.



Management

- **Identification and Removal of Trigger:**
 - Drugs, infections, or other triggers should be identified and removed if possible.
- **Supportive Care:**
 - Oral antihistamines for pruritus
 - Analgesics for pain
- **Topical Treatment:**
 - Topical corticosteroids for localized lesions
 - Emollients to keep the skin moist
- **Systemic Treatment:**
 - Systemic corticosteroids are generally reserved for severe cases and should be used cautiously.

- Antiviral therapy if herpes simplex virus is identified as a trigger.
- **Mucosal Care:**
 - Mouthwashes containing antiseptics and anesthetics for oral lesions.
 - Lubricating eye drops for ocular involvement.
- **Hospitalization:**
 - Generally not required unless the condition is severe, extensive, or complicated by other medical conditions.
- **Follow-Up:**
 - Regular dermatological assessment to monitor the resolution of lesions and any potential complications.
 - Re-evaluation of medications and other potential triggers to prevent recurrence.

Complications

- **Secondary Infections:** Risk of bacterial superinfection of skin lesions is generally low but should be monitored.
- **Scarring and Pigmentation:** Rare and generally resolves without any long-term sequelae.
- **Psychological Impact:** Generally mild but should be assessed, especially in recurrent cases.

Prognosis

- **Generally Good:** Most cases resolve without any long-term complications.
- **Recurrence:** Some individuals may experience recurrent episodes, particularly if a triggering factor is not identified and removed.

-----X-----X-----

Q: Molluscum Contagiosum

Etiology

- **Causative Agent:** Molluscum contagiosum virus (MCV), a member of the Poxviridae family.
- **Transmission:** Direct skin-to-skin contact, fomites, and autoinoculation.

- **Risk Factors:** Close physical contact, compromised immune status, and warm, humid climates.

Clinical Features

- **Skin Lesions:**
 - Dome-shaped, flesh-colored or pearly white papules.
 - Central umbilication is characteristic.
 - Size varies from 1-5 mm in diameter.
- **Distribution:**
 - Commonly affects the trunk, extremities, and genital area.
 - Rarely affects the face and mucous membranes.
- **Symptomatology:**
 - Generally asymptomatic but can be pruritic.
 - Secondary bacterial infections can occur.
- **Special Populations:**
 - In immunocompromised individuals, lesions may be more numerous and resistant to treatment.

Diagnosis

- **Clinical Examination:** Diagnosis is primarily clinical, based on the characteristic appearance of lesions.
- **Dermoscopy:** Can aid in diagnosis by showing characteristic features.
- **Histopathology:** Rarely needed, but shows intracytoplasmic inclusion bodies (molluscum bodies).

Management

- **Observation:**
 - Spontaneous resolution often occurs within 6-12 months.
- **Topical Treatments:**
 - Cantharidin
 - Imiquimod
 - Podophyllotoxin

- **Cryotherapy:**
 - Liquid nitrogen application for individual lesions.
- **Curettage or Laser Therapy:**
 - For extensive or resistant lesions.
- **Systemic Therapy:**
 - Cidofovir in severe or refractory cases, particularly in immunocompromised individuals.
- **Prevention of Spread:**
 - Avoid sharing towels, clothing, or other personal items.
 - Use barrier methods during sexual contact if genital lesions are present.

Complications

- **Secondary Infections:** Bacterial superinfection can occur, especially if lesions are scratched.
- **Scarring:** Possible but uncommon, especially if lesions are removed surgically or resolve spontaneously.

Prognosis

- **Generally Excellent:** Most cases resolve without treatment within a year.
- **Recurrence:** Possible, especially if the immune system is compromised.

Special Considerations

- **Pregnancy:** Consult a healthcare provider for appropriate management, as some treatments are contraindicated.
- **Immunocompromised Patients:** More aggressive treatment may be needed, and resolution may take longer.

-----X-----X-----

Q: Seborrheic Dermatitis in Children and Neonates

Etiology

- **Causative Factors:** Overgrowth of Malassezia yeast, increased sebum production, and genetic predisposition.
- **Age Groups Affected:** Common in neonates and adolescents.

- **Risk Factors:** Family history, hormonal changes, and environmental factors like cold, dry weather.

Clinical Features

- **Neonates:**
 - **Cradle Cap:** Yellowish, greasy scales on the scalp.
 - **Diaper Area:** Redness and scaling.
 - **Face and Body:** Erythematous patches with overlying greasy scales.
- **Children and Adolescents:**
 - **Scalp:** Dandruff-like flaking.
 - **Face:** Redness and scaling around the nose, eyebrows, and ears.
 - **Chest and Back:** Erythematous patches with mild scaling.

Diagnosis

- **Clinical Examination:** Diagnosis is primarily clinical, based on lesion morphology and distribution.
- **KOH Examination:** To rule out fungal infections.
- **Skin Biopsy:** Rarely needed, mainly to exclude other dermatoses.

Management

- **Neonates:**
 - **Mild Cleansers:** Use of mild shampoos and cleansers.
 - **Topical Emollients:** Application of mineral oil or petroleum jelly.
 - **Antifungal Creams:** Ketoconazole or clotrimazole for persistent cases.
- **Children and Adolescents:**
 - **Antidandruff Shampoos:** Selenium sulfide, zinc pyrithione, or ketoconazole.
 - **Topical Steroids:** Hydrocortisone for short-term use.
 - **Calcineurin Inhibitors:** Tacrolimus or pimecrolimus for face and intertriginous areas.
 - **Systemic Therapy:** Oral antifungals for severe cases.

Complications

- **Secondary Infections:** Risk of bacterial or fungal superinfections.
- **Skin Atrophy:** With prolonged use of topical steroids.

Prognosis

- **Neonates:** Usually resolves within the first year of life.
- **Children and Adolescents:** May persist but generally improves with age.

Special Considerations

- **Avoid Irritants:** Such as harsh soaps and fragrances.
- **Regular Follow-up:** To monitor treatment efficacy and manage complications.

-----X-----X-----

Q: Erythema Nodosum

Erythema nodosum is generally a self-limiting condition but can be a sign of an underlying systemic disease. Therefore, a thorough evaluation is essential for appropriate management.

Etiology

- **Causative Factors:** Immune response to various triggers.
- **Associated Conditions:** Infections (Streptococcal, Tuberculosis), medications (oral contraceptives, sulfonamides), systemic diseases (IBD, sarcoidosis), and malignancies.
- **Age Groups Affected:** Common in young adults, but can occur at any age.
- **Risk Factors:** Female gender, family history, and exposure to specific triggers.

Clinical Features

- **Skin Lesions:** Tender, erythematous nodules mainly on the anterior shins.
- **Systemic Symptoms:** Fever, malaise, and arthralgia.
- **Distribution:** Bilateral and symmetrical involvement of lower extremities.

Diagnosis

- **Clinical Examination:** Characteristic skin lesions.
- **Blood Tests:** Elevated ESR, CRP, and sometimes leukocytosis.
- **Skin Biopsy:** Shows septal panniculitis without vasculitis.

- **Additional Tests:** Chest X-ray, throat culture, TB tests, depending on suspected underlying condition.

Management

- **Identify Underlying Cause:** Treat the underlying condition if identified.
- **Symptomatic Relief:**
 - NSAIDs for pain and inflammation.
 - Bed rest and leg elevation.
- **Corticosteroids:** In severe or refractory cases.
- **Antibiotics:** If bacterial infection is the underlying cause.

Complications

- **Hyperpigmentation:** Residual pigmentation after lesions resolve.
- **Chronicity:** In cases associated with chronic diseases like IBD.

Prognosis

- **Self-limiting:** Most cases resolve within 3-6 weeks.
- **Recurrence:** Possible, especially if underlying condition persists.

Special Considerations

- **Regular Monitoring:** To assess treatment response and manage complications.
- **Patient Education:** About the benign nature of the condition and the importance of treating underlying diseases.

-----X-----X-----

Q: Trichotillomania

Trichotillomania, also known as trich, is when one cannot resist the urge to pull out their own hair. They may pull out the hair on their head or in other places, such as their eyebrows or eyelashes.

Etiology

- **Psychological Factors:** Often associated with stress, anxiety, or emotional trauma.

- **Neurobiological Factors:** Dysregulation of neurotransmitters like serotonin.
- **Genetic Predisposition:** Family history of obsessive-compulsive disorder (OCD) or trichotillomania.
- **Age of Onset:** Typically manifests in late childhood or early adolescence.

Clinical Features

- **Hair Pulling:** Compulsive pulling of hair from the scalp, eyebrows, eyelashes, or other body areas.
- **Bald Patches:** Varying degrees of noticeable hair loss.
- **Ritualistic Behavior:** Some individuals may inspect, play with, or ingest the pulled hairs.
- **Coexisting Conditions:** Often comorbid with anxiety disorders, depression, or OCD.

Diagnosis

- **Clinical Evaluation:** Detailed history and physical examination.
- **Psychiatric Assessment:** To rule out other psychiatric conditions.
- **Dermoscopic Examination:** To assess the condition of the scalp and hair follicles.

Management

- **Cognitive Behavioral Therapy (CBT):** The most effective psychological treatment.
- **Pharmacotherapy:**
 - SSRIs like fluoxetine or clomipramine for adults.
 - N-acetylcysteine has shown promise in some studies.
- **Habit Reversal Training:** Techniques to recognize triggers and adopt alternative behaviors.
- **Psychoeducation:** Educating the patient and family about the condition.

Complications

- **Skin Infections:** Secondary to open sores or ingrown hairs.
- **Psychological Distress:** Low self-esteem, social withdrawal, and academic or occupational impairment.
- **Gastrointestinal Issues:** If hair is ingested, leading to trichobezoars.

Prognosis

- **Variable Course:** Can be chronic and difficult to treat in some cases.
- **Better Prognosis:** When treated early and comprehensively.

Special Considerations

- **Multidisciplinary Approach:** Often requires a team of healthcare providers including dermatologists, psychiatrists, and psychologists.
- **Family Involvement:** Crucial for treatment adherence and emotional support.

-----X-----X-----

Q: Pediculosis

Etiology

- **Causative Agent:** *Pediculus humanus capitis* (head lice), *Pediculus humanus corporis* (body lice), *Pthirus pubis* (pubic lice).
- **Transmission:** Direct contact, sharing personal items like combs, hats, or clothing.
- **Risk Factors:** Overcrowding, poor hygiene, close contact settings like schools.

Clinical Features

- **Pruritus:** Intense itching due to allergic reaction to lice bites.
- **Erythema:** Redness and inflammation at bite sites.
- **Nits:** Lice eggs attached to hair shafts.
- **Secondary Infections:** Possible bacterial infections due to scratching.

Diagnosis

- **Visual Inspection:** Identification of live lice or nits.
- **Dermoscopy:** To differentiate between nits and other particles like dandruff.
- **Skin Scraping:** Rarely used, mostly for body lice.

Management

- **Topical Insecticides:**
 - Permethrin 1% cream rinse

- Malathion 0.5% lotion
- **Oral Treatment:** Ivermectin in cases resistant to topical treatment.
- **Mechanical Removal:** Nit combing.
- **Environmental Measures:** Washing clothing and bed linens in hot water.

Complications

- **Skin Infections:** Impetigo, cellulitis due to secondary bacterial infection.
- **Psychological Impact:** Social stigma, anxiety, and embarrassment.
- **Treatment Resistance:** Increasing resistance to over-the-counter pediculicides.

Prognosis

- **Excellent:** With appropriate treatment.
- **Recurrence:** Possible if the source of infestation is not eliminated.

Special Considerations

- **School Policies:** Vary widely; some require "no-nit" policies, while others are more lenient.
- **Public Health Measures:** Education and awareness to prevent outbreaks.

-----X-----X-----

Q: Acne in Adolescence

Etiology

- **Hormonal Fluctuations:** Increased androgens during puberty.
- **Sebum Overproduction:** Excessive oil secretion.
- **Clogged Pores:** Accumulation of dead skin cells.
- **Bacterial Infection:** Propionibacterium acnes colonization.
- **Genetic Predisposition:** Family history of acne.

Clinical Features

- **Comedones:** Open (blackheads) and closed (whiteheads).
- **Papules:** Small, red, inflamed bumps.

- **Pustules:** Pus-filled lesions.
- **Nodules:** Large, painful lumps beneath the surface of the skin.
- **Cysts:** Large, pus-filled lumps deeper in the skin.

Diagnosis

- **Clinical Examination:** Visual inspection of affected areas.
- **Severity Grading:** Mild, moderate, and severe based on lesion type and distribution.
- **Differential Diagnosis:** Rosacea, folliculitis, keratosis pilaris.

Management

- **Topical Treatments:**
 - Benzoyl peroxide
 - Retinoids (tretinoin, adapalene)
 - Topical antibiotics (clindamycin)
- **Oral Treatments:**
 - Antibiotics (doxycycline, minocycline)
 - Oral contraceptives for females
 - Isotretinoin for severe cases
- **Adjunctive Therapies:**
 - Chemical peels
 - Laser therapy
- **Skin Care Regimen:** Gentle cleansing, oil-free moisturizers, non-comedogenic products.

Complications

- **Scarring:** Atrophic, hypertrophic, or keloidal.
- **Hyperpigmentation:** Post-inflammatory changes.
- **Psychological Impact:** Reduced self-esteem, depression, social withdrawal.

Prognosis

- **Variable:** Depends on severity and treatment response.

- **Long-term Management:** Often required for persistent or cystic acne.

Special Considerations

- **Isotretinoin:** Requires strict monitoring due to potential side effects like liver dysfunction and teratogenicity.
- **Diet and Lifestyle:** Limited evidence linking diet to acne; however, a balanced diet and stress management may help.

Management Strategy	Description	Medications/Procedures Involved	Duration & Frequency	Side Effects & Precautions
Topical Retinoids	First-line treatment for mild to moderate acne. Helps unclog pores.	Tretinoin, Adapalene, Tazarotene	Daily application	Skin irritation, photosensitivity
Topical Antibiotics	Used to reduce inflammation and bacterial growth.	Clindamycin, Erythromycin	1-2 times daily	Antibiotic resistance, skin irritation
Benzoyl Peroxide	Antimicrobial agent effective against P. acnes bacteria.	Over-the-counter creams and gels	Daily application	Skin dryness, bleaching of fabric
Oral Antibiotics	For moderate to severe acne, or acne that covers large areas.	Doxycycline, Minocycline, Tetracycline	Short-term (3-4 months)	GI upset, antibiotic resistance
Hormonal Therapy	For females with acne exacerbated	Birth control pills, Spironolactone	Monthly cycles	Weight gain, blood clots

	by hormonal fluctuations.			
Oral Retinoids (Isotretinoin)	For severe or treatment-resistant acne.	Isotretinoin (Accutane)	4-6 months	Dry skin, elevated lipids, teratogenicity
Chemical Peels	Exfoliates the skin and helps in unclogging pores.	Salicylic acid, Glycolic acid	Monthly sessions	Redness, peeling
Light and Laser Therapy	Targets P. acnes and reduces oil gland size.	Blue light, Pulsed dye laser	Multiple sessions	Temporary redness, potential scarring
Corticosteroid Injection	For large, painful, treatment-resistant nodules.	Intralesional corticosteroid	As needed	Skin thinning, hypopigmentation
Comedone Extraction	Physical removal of blackheads and whiteheads.	Sterile needle or loop extractor	As needed	Scarring, infection
Supportive Care	Adjunctive measures to improve skin health.	Oil-free moisturizers, non-comedogenic products	Daily	Varies depending on the product

-----X-----X-----

Q: Treatment of acne vulgaris

- ✚ Treatment includes a combination of topical and systemic therapies, lifestyle modifications, and addressing the psychosocial aspects of the condition.

1. Pathophysiology Understanding:

- Acne vulgaris is a multifactorial skin condition primarily affecting pilosebaceous units.
- Key factors include sebum overproduction, follicular hyperkeratinization, Propionibacterium acnes (P. acnes) proliferation, and inflammation.
- Hormonal influences, particularly androgens, play a significant role in sebum production.

2. Clinical Presentation:

- Characterized by non-inflammatory lesions (open and closed comedones) and inflammatory lesions (papules, pustules, nodules).
- Commonly affects areas with a high density of sebaceous glands: face, back, and chest.
- Severity varies from mild comedonal acne to severe nodulocystic acne.

3. Treatment Principles:

- Tailored to acne severity and individual patient factors.
- Aims to reduce sebum production, prevent follicular plugging, limit bacterial growth, and reduce inflammation.
- Requires patient education on realistic expectations and adherence to treatment.

4. Topical Therapies:

- Retinoids (e.g., tretinoin, adapalene): Normalize follicular epithelial desquamation, anti-inflammatory.
- Benzoyl Peroxide: Antimicrobial, comedolytic, and mild anti-inflammatory properties.
- Antibiotics (e.g., clindamycin, erythromycin): Target P. acnes, used in combination to reduce antibiotic resistance.
- Azelaic Acid: Antimicrobial, comedolytic, and anti-inflammatory, useful in post-inflammatory hyperpigmentation.
- Salicylic Acid: Keratolytic agent, assists in unclogging pores.

5. Systemic Therapies:

- Antibiotics (e.g., doxycycline, minocycline): Used for moderate to severe inflammatory acne, risk of antibiotic resistance.
- Hormonal Therapies (e.g., oral contraceptives, spironolactone): Effective in females with hormonal acne.
- Isotretinoin: For severe, scarring, or treatment-resistant acne; regulates sebum production, anti-inflammatory, alters follicular keratinization.
- Consideration of patient's overall health, potential side effects, and contraindications is crucial.

6. Physical and Adjunctive Therapies:

- Light and Laser Therapy: Targets P. acnes, reduces inflammation, and promotes collagen remodeling.
- Chemical Peels: Use of acids (e.g., glycolic acid, salicylic acid) to exfoliate the skin, reduce comedones.
- Extraction of Comedones: Performed by professionals to reduce risk of scarring and infection.

7. Lifestyle and Dietary Factors:

- Evidence suggests a link between diet and acne, particularly high glycemic index foods and dairy.
- Stress management and avoidance of acne triggers (e.g., certain cosmetics) are recommended.

8. Psychosocial Impact:

- Acne can significantly impact quality of life, self-esteem, and mental health.
- Addressing psychological aspects is an integral part of acne management.

9. Emerging Treatments and Research:

- Newer retinoids, novel anti-inflammatory agents, and vaccine development against P. acnes.
- Microbiome research and personalized medicine approaches are gaining interest.

10. Treatment Monitoring and Follow-up:

- Regular follow-up to assess treatment efficacy, side effects, and patient adherence.
- Adjustments to treatment regimens based on response and emerging issues.

11. Prevention and Maintenance:

- Continuation of topical therapies post-clearance to prevent recurrence.
- Education on skin care routines and avoidance of comedogenic products.

12. Challenges and Considerations:

- Antibiotic resistance in long-term antibiotic therapy.
- Teratogenic risk of isotretinoin, requiring strict pregnancy prevention measures.
- Management of post-acne scarring and hyperpigmentation.

13. Patient Education and Engagement:

- Importance of adherence to treatment regimens.
- Understanding of acne pathophysiology to set realistic expectations.
- Awareness of potential side effects and when to seek medical advice.

Acne Vulgaris	Details
Pathophysiology	Multifactorial skin condition affecting pilosebaceous units. Involves sebum overproduction, follicular hyperkeratinization, P. acnes proliferation, inflammation. Hormonal influences, especially androgens, are significant.
Clinical Presentation	Non-inflammatory (comedones) and inflammatory lesions (papules, pustules, nodules). Affects areas with dense sebaceous glands: face, back, chest. Severity ranges from mild to severe nodulocystic acne.
Treatment Principles	Tailored to severity and patient factors. Aims to reduce sebum, prevent follicular plugging, limit bacterial growth, reduce inflammation. Requires patient education and adherence.

Topical Therapies	Retinoids (e.g., tretinoin, adapalene). Benzoyl Peroxide. Antibiotics (e.g., clindamycin, erythromycin). Azelaic Acid. Salicylic Acid.
Systemic Therapies	Antibiotics (e.g., doxycycline, minocycline). Hormonal Therapies (e.g., oral contraceptives, spironolactone). Isotretinoin. Consideration of side effects and contraindications.
Physical Therapies	Light and Laser Therapy. Chemical Peels. Extraction of Comedones.
Lifestyle and Diet	Link between diet (high glycemic index foods, dairy) and acne. Stress management and avoidance of triggers.
Psychosocial Impact	Significant impact on quality of life, self-esteem, mental health. Integral part of management.
Emerging Treatments	Newer retinoids, anti-inflammatory agents, vaccine against P. acnes. Microbiome research, personalized medicine.
Monitoring and Follow-up	Regular assessment of treatment efficacy, side effects, adherence. Adjustments based on response.
Prevention and Maintenance	Continuation of topical therapies. Education on skin care and avoidance of comedogenic products.
Challenges	Antibiotic resistance. Teratogenic risk of isotretinoin. Management of post-acne scarring and hyperpigmentation.
Patient Education	Importance of treatment adherence. Understanding of acne pathophysiology. Awareness of side effects and medical advice seeking.

-----X-----X-----

Q: Psychological Complications of Acne in Children

Self-Esteem and Self-Image Issues

- **Negative Self-Perception:** Children may view themselves as less attractive due to acne.

Social Withdrawal and Isolation

- **Avoidance of Social Activities:** Reluctance to participate in social events or group activities.
- **School Absenteeism:** Avoiding school to escape teasing or bullying.

Anxiety Disorders

- **Social Anxiety:** Fear of negative evaluation in social settings.
- **Generalized Anxiety:** Excessive worry about acne and its consequences.

Depression

- **Mood Swings:** Frequent emotional ups and downs.
- **Persistent Sadness:** Ongoing feelings of unhappiness or hopelessness.

Academic Impact

- **Reduced Concentration:** Difficulty focusing on studies due to preoccupation with acne.
- **Lower Academic Performance:** Potential decline in grades.

Interpersonal Difficulties

- **Strained Relationships:** Tension in friendships and family relationships due to irritability or withdrawal.

Body Dysmorphic Disorder (BDD)

- **Obsession with Flaws:** Excessive preoccupation with perceived defects, which may be minor but are magnified in the child's mind.

Suicidal Ideation

- **Thoughts of Self-Harm:** In extreme cases, persistent depression and anxiety may lead to suicidal thoughts.

Treatment-Related Stress

- **Anxiety about Treatment Outcome:** Worry about the effectiveness of acne treatments.
- **Stress from Side Effects:** Anxiety about potential side effects of medications like isotretinoin.

Coping Mechanisms

- **Negative Coping:** Unhealthy coping strategies like substance abuse.

Long-term Impact

- **Chronic Psychological Issues:** If not addressed, the psychological impact can extend into adulthood.

---X-----X---



BONE AND JOINT DISORDERS

Q: Approach to a child with Talipes equinovarus at different ages (CTEV)

- ✚ The treatment plan should be individualized based on the severity of the deformity and the response to treatment.
- ✚ Family education and support are integral parts of the management plan.
- ✚ Lifelong follow-up may be necessary in some cases to monitor for recurrence or complications.

Age Group	Initial Assessment	Treatment Modalities	Follow-up and Monitoring	Additional Notes
Newborn 3 months	Physical examination Radiographic assessment	Ponseti method Serial casting Achilles tenotomy if needed	Weekly casting Monitoring for skin irritation or complications	Early intervention is crucial for optimal outcomes.
3 months 1 year	Re-assessment of deformity Radiographic assessment if needed	Continuation or modification of Ponseti method Surgical intervention if casting fails	Monthly to quarterly visits Monitoring for recurrence	Foot abduction braces are often used after successful casting.
1 year 3 years	Assessment of gait Radiographic assessment	Surgical intervention for residual or recurrent deformities Physical therapy	Quarterly to bi-annual visits Gait analysis	Footwear modification may be needed.
3 years 6 years	Gait analysis Radiographic assessment	Surgical intervention for residual deformities Physical therapy	Bi-annual visits Monitoring for complications of surgery	Social and psychological support for school integration.
6 years and above	Detailed musculoskeletal and functional assessment	Surgical interventions for complications or residual deformities Physical therapy	Annual visits Long-term monitoring for foot and ankle function	May require adaptive devices for mobility.

Initial Assessment

- **Physical Examination:** Assess foot deformity, flexibility, and presence of other congenital anomalies.
- **Family History:** Evaluate for genetic factors, as CTEV can have a hereditary component.
- **Ultrasound or X-Ray:** Imaging is used in atypical cases or older children to assess bone structure.

Non-Surgical Treatment

1. **Ponseti Method:**

- **Manipulation and Casting:** Begins shortly after birth. Weekly gentle manipulation and casting to gradually correct the foot position.
- **Achilles Tenotomy:** Often required in about 90% of cases to correct equinus deformity.
- **Bracing:** After correction, foot abduction braces are used to maintain correction. Initially full-time, then only during naps and night-time up to 4 years of age.

2. **French Functional (Physical Therapy) Method:**

- Daily stretching, mobilization, and taping.
- Requires active participation from parents/caregivers.
- Used as an alternative or adjunct to the Ponseti method.

Surgical Treatment

- **Indications:** Reserved for cases where non-surgical methods fail or in older children with neglected CTEV.
- **Types of Surgery:**
 - Soft tissue release (posterior, medial, and sometimes lateral release).
 - Osteotomies for severe deformities or older children.
 - Tendon transfers for dynamic deformities or recurrences.

Post-Treatment Monitoring

- **Regular Follow-Up:** To monitor for recurrence, which is common in CTEV.

- **Physical Examination:** Assess foot alignment and flexibility.
- **Parental Education:** On the importance of brace compliance and monitoring for signs of recurrence.

Complications and Management

- **Recurrence:** Managed with repeat casting, possible surgery.
- **Brace Issues:** Skin irritation, poor fit; requires regular brace adjustment.
- **Residual Deformity:** May require further treatment or surgery.

-----X-----X-----

Q: Congenital Dislocation of Hip / DDH; Describe developmental dysplasia of the hip (DDH). Enumerate risk factors for the same. Enlist its clinical features. How do you confirm its diagnosis? Outline the management.

Developmental Dysplasia of the Hip (DDH)

Developmental Dysplasia of the Hip (DDH) is a spectrum of congenital and acquired conditions affecting the hip joint's anatomy and function. It ranges from mild instability to complete dislocation of the hip. The condition can lead to long-term complications like osteoarthritis if not adequately managed.

Risk Factors

- Female sex
- Firstborn child
- Family history of DDH
- Breech presentation
- Oligohydramnios
- Swaddling with legs extended and pressed together

Clinical Features

- Asymmetry of the gluteal or thigh folds
- Limited range of motion in one hip
- Positive Ortolani or Barlow test in infants
- Limping or waddling gait in older children
- Leg-length discrepancy

- Delayed walking or abnormal gait

Diagnosis

- **Physical Examination:** Ortolani and Barlow tests in newborns
- **Ultrasound:** Recommended for infants under 6 months
- **X-ray:** Useful for children over 6 months and for confirming findings in older children
- **MRI/CT:** Rarely used, but can provide detailed images

Management

- **Newborn - 6 weeks:** Pavlik harness for maintaining hip in flexion and abduction
- **6 weeks - 6 months:** Closed reduction followed by Spica casting if Pavlik harness is unsuccessful
- **6 months - 18 months:** Open reduction and Spica casting, possible pelvic osteotomy
- **18 months - 5 years:** Open reduction, femoral and/or pelvic osteotomy
- **5 years and above:** Complex surgical procedures like salvage osteotomies, arthroplasty in some cases

Follow-up: Regular monitoring through physical exams and imaging studies is crucial at all stages.

Age Group	Initial Assessment	Treatment Modalities	Follow-up and Monitoring	Additional Notes
Newborn 6 weeks	Physical examination (Barlow & Ortolani tests) Ultrasound if positive findings	Pavlik harness Closed reduction for failed Pavlik harness	Weekly to bi-weekly visits Ultrasound monitoring	Early diagnosis and treatment are crucial.
6 weeks 6 months	Physical examination Ultrasound or X-ray	Closed reduction Spica casting	Monthly visits Radiographic monitoring	Treatment becomes more challenging as the child grows.

6 months 18 months	Physical examination X-ray	Open reduction Spica casting or surgery	Quarterly visits Radiographic monitoring	Surgical intervention is often needed.
18 months 5 years	Physical examination X-ray	Open reduction Pelvic osteotomy	Bi-annual visits Radiographic monitoring	More complex surgical procedures may be required.
5 years and above	Physical examination X-ray	Open reduction Femoral and/or pelvic osteotomy	Annual visits Long-term monitoring for hip function	Treatment at this stage is complex and may have less optimal outcomes.

-----X-----X-----

Q: Definition of Skeletal Dysplasia

Skeletal dysplasias are a heterogeneous group of more than 400 disorders characterized by abnormalities in the size and shape of the limbs, trunk, and skull. These conditions are generally caused by mutations in genes that affect cartilage and bone development.

Table of Skeletal Dysplasias

Disorder	Etiology	Clinical Features	Diagnosis	Management
Achondroplasia	FGFR3 gene mutation	Short stature, macrocephaly, limb deformities	Clinical, X-ray, Genetic testing	Multidisciplinary care
Osteogenesis Imperfecta	COL1A1, COL1A2 gene mutations	Fragile bones, blue sclera, hearing loss	Clinical, X-ray, Genetic testing	Bisphosphonates, physical therapy
Thanatophoric Dysplasia	FGFR3 gene mutation	Extremely short limbs, narrow chest	Prenatal ultrasound, X-ray	Supportive, usually fatal

Hypochondroplasia	FGFR3 gene mutation	Mild short stature, stocky build	Clinical, X-ray	Growth hormone therapy
Spondyloepiphyseal Dysplasia	COL2A1 gene mutation	Short trunk, cleft palate, myopia	Clinical, X-ray	Orthopedic intervention
Diastrophic Dysplasia	SLC26A2 gene mutation	Short stature, joint deformities, hitchhiker thumb	Clinical, X-ray	Surgery, physical therapy
Campomelic Dysplasia	SOX9 gene mutation	Bowed limbs, narrow chest, ambiguous genitalia	Prenatal ultrasound, X-ray	Supportive, often fatal
Pseudoachondroplasia	COMP gene mutation	Short stature, normal facial features, joint laxity	Clinical, X-ray	Physical therapy, orthopedic surgery
Cleidocranial Dysplasia	RUNX2 gene mutation	Absent or partial collarbones, delayed closure of fontanelles	Clinical, X-ray	Dental and orthopedic intervention
Chondrodysplasia Punctata	Multiple genes	Stippled epiphyses, cataracts, short stature	Clinical, X-ray	Supportive care

-----X-----X-----

Q: Achondroplasia

Achondroplasia is the most common form of short-limbed dwarfism, characterized by abnormal bone growth that results in short stature with disproportionately short arms and legs, a large head, and characteristic facial features.

Etiology

- **Genetic Mutation:** Most commonly due to a mutation in the FGFR3 gene.
- **Sporadic Occurrence:** About 80% of cases occur spontaneously due to new mutations.
- **Inheritance:** Autosomal dominant pattern, although most cases result from new mutations.

Clinical Features

- **Short Stature:** Disproportionate dwarfism
- **Macrocephaly:** Large head with frontal bossing
- **Limb Deformities:** Short arms and legs, particularly the upper arms and thighs
- **Spinal Deformities:** Lumbar lordosis, possible kyphosis
- **Facial Features:** Depressed nasal bridge, protruding jaw
- **Limited Range of Motion:** Especially at the elbows
- **Hand Anomalies:** Trident hand, short fingers
- **Delayed Milestones:** Motor development may be delayed due to limb and spinal issues

Diagnosis

- **Prenatal Ultrasound:** May show signs as early as the second trimester
- **Physical Examination:** Characteristic physical features
- **X-ray:** Shows abnormal bone growth and structure
- **Genetic Testing:** Confirmatory, especially if there's a family history

Management

- **Growth Hormone Therapy:** Controversial, limited effectiveness
- **Orthopedic Interventions:** Limb-lengthening surgeries, correction of bowed legs
- **Physical Therapy:** To improve mobility and muscle strength
- **Neurological Monitoring:** Due to risk of spinal cord compression
- **ENT Consults:** For frequent ear infections
- **Supportive Care:** Adaptive devices, occupational therapy
- **Psychological Support:** For both the child and family

Q: Classification and management of Osteogenesis Imperfecta

Classification of Osteogenesis Imperfecta

Type	Clinical Features	Severity	Genetic Mutation
Type I	Mild, blue sclera, minimal deformity	Mild	COL1A1, COL1A2
Type II	Perinatal lethal, severe deformities	Severe	COL1A1, COL1A2
Type III	Progressively deforming, fractures common	Severe	COL1A1, COL1A2
Type IV	Moderate severity, normal sclera	Moderate	COL1A1, COL1A2
Type V	Similar to Type IV but with calcification between forearm bones	Moderate	IFITM5
Type VI	Moderate severity, distinct histological features	Moderate	SERPINF1
Type VII	Severe to moderate, rhizomelia	Moderate to Severe	CRTAP
Type VIII	Severe to moderate, white sclera	Moderate to Severe	LEPRE1
Type IX	Moderate severity, normal sclera	Moderate	PPIB
Type X	Moderate severity, normal sclera	Moderate	SERPINH1

Management of Osteogenesis Imperfecta

Medical Management

- **Bisphosphonates:** Used to increase bone density and reduce fractures. Commonly used drugs include Pamidronate, Alendronate, and Zoledronic acid.
- **Calcium and Vitamin D Supplementation:** To support bone health.
- **Growth Hormone Therapy:** Investigational, may help in increasing height and improving bone density.
- **Anti-sclerostin Antibodies:** Investigational, may improve bone density and strength.

Surgical Management

- **Intramedullary Rodding:** Surgical insertion of metal rods into the long bones to provide stability and reduce fractures.
- **Spinal Fusion:** For severe scoliosis.
- **Corrective Osteotomy:** To correct bone deformities.

Supportive Management

- **Physical Therapy:** To improve mobility and muscle strength.
- **Mobility Aids:** Wheelchairs, braces, and other aids as needed.
- **Pain Management:** Analgesics and other medications to manage chronic pain.
- **Dental Care:** Due to the high prevalence of dental issues in OI, regular dental check-ups are advised.

Monitoring

- **Regular X-rays:** To monitor bone health and the effectiveness of treatment.
- **DEXA Scans:** To monitor bone density.
- **Pulmonary Function Tests:** Especially in severe cases with chest deformities.

Genetic Counseling

- **Prenatal Testing:** For families with a history of OI.
- **Family Planning:** Discussion about the risks of inheritance.

Psychological Support

- **Counseling:** For the child and family to cope with the emotional burden of the disease.

Respiratory Support

- **Oxygen Therapy:** In severe cases with respiratory compromise.

Fracture Care

- Prompt and appropriate treatment of fractures including immobilization and surgical intervention as needed.

Management Strategy	Type I	Type II	Type III	Type IV	Type V-X
Bisphosphonates	✓	X	✓	✓	✓

Physical Therapy	✓	X	✓	✓	✓
Surgical Interventions (Intramedullary Rodding)	✓	X	✓	✓	✓
Calcium and Vitamin D Supplementation	✓	X	✓	✓	✓
Mobility Aids	✓	X	✓	✓	✓
Genetic Counseling	✓	✓	✓	✓	✓
Respiratory Support	X	✓	✓	X	X
Pain Management	✓	X	✓	✓	✓
Dental Care	✓	X	✓	✓	✓
Fracture Care	✓	X	✓	✓	✓

-----X-----X-----

